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FEVERS OF THE MACKAY DISTRICT, QUEENSLAND.

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The Mackay District.

THE Mackay district presents several points of importance in relation to the study of its fevers.

Firstly, it is important to know definitely if scrub typhus is present here. If so, the Mackay district becomes, on present knowledge, the most southern outpost of this disease in the world, and so offers a site for the study of the factors which limit its distribution.

Secondly, in this district there occurred some years ago a dramatic outbreak of a fever of undetermined nature, which became known as "Sarina fever". This calls for clarification.

Thirdly, the Mackay district (together with the Proserpine district) forms a clearly demarcated climatic subregion which merits ecological investigation in its own right. It is situated nearly half-way up the east coast of Queensland. Here the curve of the Connors and Clarke Ranges, rising to 4190 feet at Mount Dalrymple, encloses a wide area of fertile country drained by the Pioneer River. Its latitude of 21° south places it within the tropics. It enjoys an abundant rainfall averaging about 60 inches annually, most of which falls in the first three months of the year. These months are hot and humid; the winter climate is pleasant. Originally it was clothed in part with tropical rain forest, commonly called "scrub", in part with open forest and tall grass. Much has been cleared for cultivation. This well-watered region, in which one would expect to find types of disease and disease vectors which are favoured by heat and humidity, is circumscribed on north, west and south by drier belts, and is thereby separated widely from the other high-rainfall areas which lie along the Queensland coast to the north and south (Figure I). While the fevers of the latter areas have frequently been described, only one report, that by Wheatland (1924) on "Sarina fever", has been published on fevers endemic in the Mackay district.

Pastoral settlement began on the Pioneer River in 1862, two years after its discovery by John Mackay. Sugarcane was planted in 1865 on the river flats, and this industry rapidly extended to become locally all-important and to make Mackay the leading centre of sugar production in Queensland. In 1952, 125,000 acres were assigned to cane-growing, and about 190,000 tons of sugar were produced by the seven mills in the area. There are over two hundred

dairy farms, particularly on the ranges, and tropical fruits and vegetables are grown. Mackay is also the centre for a large area of pastoral country further inland. The city of Mackay has a population of 15,000 and the neighbouring shires have a further 22,500.

It should not be inferred from our interest in fevers that Mackay is unduly unhealthy. The average death rate over the last five published years for the Mackay statistical district (which does not differ greatly from the area shown in Figure II, except that the area around Proserpine is included) is 7·2 per thousand—the lowest among the statistical districts of Queensland.

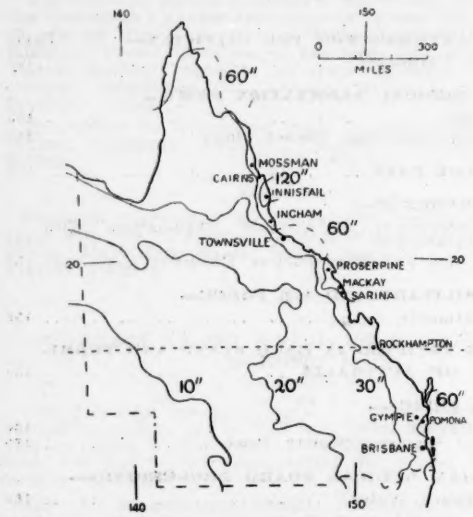

FIGURE I.

Map of Queensland showing the situation of Mackay and how the high rainfall area surrounding it, delineated by the 60" isohyet, is separated by drier belts from the high rainfall areas to the north and south.

Past Records of Fever.

The story of fevers in the Mackay district begins with the original expedition of John Mackay in 1860 (Roth, 1908). On May 22, six days after crossing the Clarke Range into the basin of the Pioneer River, and while camped on the bank of the river, two of the party became ill with severe headaches and inability to eat. "Supposing it to be the primary symptoms of fever and ague, small doses of quinine were administered night and morning", but without improvement. Three days later another fell ill, and after two more days a fourth. One patient, Duke, an aboriginal from New South Wales, died on the twenty-third day of illness; the other three recovered after a prostrating illness that lasted three or four weeks.

The incapacitation of four of the six members of the party not only was distressing in itself, but also placed the young leader—he was but twenty-one—in a serious predicament. Stores ran low. Each day's delay lessened the chances of the sick, and also the well, reaching civilization 150 miles distant. Yet during that month of illness there were only occasional days on which they could even attempt to struggle homeward. The sudden death of Duke on one of the days of travel augmented the despondency. Mackay, writing years afterwards, tells how the succeeding few days "stand out through the long vista of years in a bolder and more harrowing relief than any other incident of an eventful life". The two who fortunately remained well—Mackay and Macrossan—tended the sick, caught fish and shot birds for them and made soup when they were unable to eat solid food. With delight a pot of Liebig's essence of meat was discovered in a saddlebag, and from it "tea" was made. By June 19, the invalids were so far recovered that they could resume the journey back to Rockhampton, but a few days later one became delirious and this enforced another week's spell. A diary entry on June 30 records great improvement in all hands.

What was the fever that attacked them? Clinical details on which to make a diagnosis are scanty. The symptoms mentioned are severe headache, anorexia, pain in the back, delirium and inability to dress or to mount a horse. The fever was not malaria, as was thought at first, for it was continuous and prolonged and did not respond to quinine. It could hardly have been typhoid, for the onset was acute and the chance of meeting *Salmonella typhosa* in the wilds

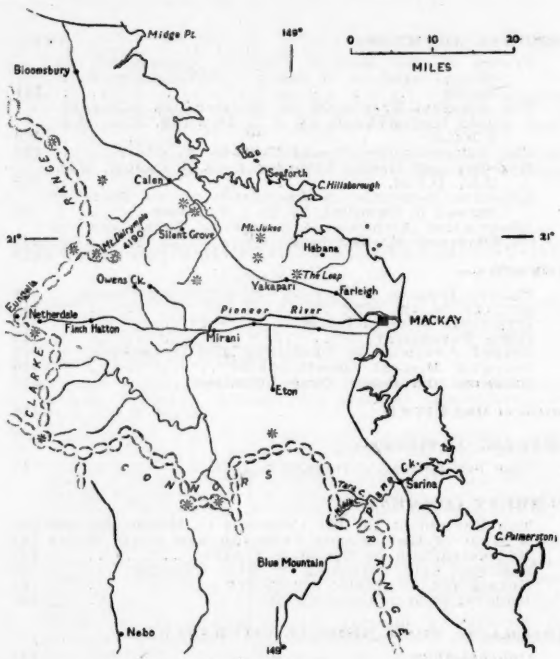

FIGURE II.

Map of the Mackay district.

remote; nor was a carrier in the party likely—they had already been camping together for four months. On the whole, scrub typhus seems to be the most likely diagnosis.

Hardly had his mates recovered when it was Mackay's turn to suffer from fever. This was of short duration and obscure nature and did not interrupt the progress of the journey.

> June 30th. . . . Towards sundown I felt the first symptoms of fever, in the shape of an intense headache, followed by such a severe fit of the shakes as to make riding a difficult matter. We camped on a small plain surrounded by scrub, and there being no water, tied our horses up. The horrors of that night I shall never forget, the fever producing a burning thirst; and our evening meal having exhausted our supply of water I was unable to quench it. During the night a heavy dew had fallen, and while the others were packing up I crawled, more than walked, to a small gully, which, following for about 100 yards in the scrub, I found about a pint of muddy water, which, from the dead leaves, had a strong, pungent taste—to me it was nectar.
>
> July 1st. Travelled today. . . . During the day I felt better, though still very weak.
>
> July 2nd. Started early. . . . At noon I was again attacked with fever, but managed to ride along.

Presumably that was the end of the attack. At least it received no further mention in Roth's extract from Mackay's diary.

The experiences that befell his party could not have been uncommon, for in 1882 Mackay wrote of the grateful memories "many of the old fever stricken pioneers" must have retained for the kind attentions of Mrs. Ready—the first white woman in the district.

Precise records of these "fever stricken pioneers" are scanty. In July, 1873, the Mackay Cemetery was opened and the first burial was of a man, aged twenty-eight years, from Meadowlands (just west of Mackay) who died of "fever". Of the 282 burials in the first five and a half years (1873 to 1878), two deaths were ascribed to "intermittent fever", one to "typhoid fever" and 21 simply to "fever". As an index of infant mortality, it may be noted that 110 of these burials were of infants under two years of age. Only one infant died of "fever".

An important episode in the history of Mackay was the importation of South Sea Island labour. This began in 1866 or 1867 and was carried on steadily as the sugar industry expanded. It was early subjected to controls and restrictions because of abuses and was finally abolished in 1904. The introduction of thousands of Islanders over a period of nearly forty years must have had some effect on the incidence of infectious diseases in the district, but we cannot offer any comment as to its extent.

Plague was an unwelcome visitor to Mackay in 1908 and 1909, and caused three deaths. Pandemic influenza invaded the district between May and August, 1919.

In the first two decades of the present century, typhoid was common in Mackay (as in Queensland generally) and was responsible for a number of deaths each year. It was liable to break out at one of the mill centres. It practically disappeared after earnest attention was given to sanitation. Only six cases have been notified from the district in the last twenty-five years, the last being in 1941. A carrier was, however, detected in 1946.

"Sarina Fever".

From 1915 onward a series of cases of fever drew attention to West Plane Creek. By West Plane Creek is meant that area lying to the westward of the town of Sarina (the original name of which was Plane Creek) and extending to the Connors Range, a distance of about eight miles. The area is traversed by Plane Creek and its tributaries, Middle and Tara Creeks. Flat areas, mostly under cultivation with sugarcane, border these creeks, and low hills separate them. They end in steep, scrub-covered gullies running into the range. The area was opened up for sugar-farming shortly before 1896, in which year the Plane Creek mill made its first crushing. Its population at the relevant period was about 150.

Occasional fatal cases of fever had occurred previously at West Plane Creek; there was one such in 1904. In the wet season of 1915-1916, at least four breadwinners in this small community died from fever, and this aroused alarm. Cases, some of which were fatal, recurred in subsequent years. Every farm but two of the dozen situated at the head of Plane Creek was affected, and there were cases also on Middle and Tara Creeks. In one family there were six cases in the course of three successive wet seasons. We have not been able to find a full record of the incidence, but Dr. R. W. Telford, State Health Officer, mentioned in June, 1922, that the cases he had then investigated numbered about 53, with 10 deaths. This was before his survey was completed. He expected that there were a great many more cases. His final report is not available, but from other information we consider that his provisional totals might be about doubled. An independent estimate is that there were 19 deaths from 1915 to 1922.

Telford's estimate was quoted by the local member, E. B. Swayne, in an account of the outbreak he gave to the Queensland Parliament in August, 1922. Swayne quoted also the following notes furnished to him by Dr. Stuart Kay, the Health Officer for Sarina Shire:

(1) Men, agricultural workers, are mostly affected by it.

(2) Cases: generally those engaged in opening up new land.

(3) Locality: in elevated part of district from which the water runs through a more populated area, and it is only in the upper part that cases have occurred.

(4) Infected persons have almost all drunk only tank water.

(5) Flies are not bad.

(6) Persons who have had recent inoculation with T.A.B. are rarely infected.

(7) Fever is of a typhoid type.

(8) All cases have been notified as typhoid and treated as such, specimens being sent to the Government laboratories. All results have been negative.

Wheatland (1924) gave further epidemiological notes as well as a clinical description. The cases occurred from November to April—that is, during the hot, wet season. The men who were affected were engaged in clearing and ploughing new ground and ground which had been cleared and again covered with secondary growth, particularly lantana. It may be noted that activity in clearing was routinely associated with the first few months of the year before cane crushing or planting started.

Several old residents said there were no cases of fever at the time the original rain forest was first cleared. One resident associated infection with the edges of the "scrub" rather than the "scrub" itself. The last death in the series, in May, 1922, was that of the surveyor who surveyed a route through lantana and "scrub" for the road over the range at the head of Plane Creek.

The principal clinical features, as described by Wheatland, included an acute onset, shivers and prolonged and sometimes severe post-orbital headache. The fever had a duration like that of typhoid or somewhat shorter and declined by lysis. A mottled rash all over the body but more marked on chest and abdomen was present in an unspecified proportion of cases. The lymph nodes might be enlarged. The bowels were usually normal, distension was uncommon, the spleen was seldom palpable. Some cases were mild and did not show the full range of symptoms mentioned; others were very severe with toxæmia, delirium, cyanosis, "septic rashes", diarrhœa and perhaps a fatal termination from heart failure as early as the second week. The mortality was about 20% to 30%—a rather higher figure than the 19% given by Telford.

One patient, interviewed twenty-six years afterwards, stated that at the time of his illness he developed wasting of the small muscles of the hands. The right hand recovered, but the muscles of the left hand never regained their former size or strength. There had also been a loss of sensation in the left hand, which lasted twelve months.

There are no autopsy records.

Not only did the cases of fever at West Plane Creek cause concern to the populace, but also the diagnosis caused cogitation among the doctors. In the years when the fever first came to notice, typhoid was prevalent in the Mackay district and was naturally the first thought. Occasional cases may, indeed, have been typhoid, but in general the clinical features and epidemiology showed differences. The terms "atypical typhoid" and "paratyphoid" and, later, "Sarina typhoid" came into use. At least an enteric hypothesis gave a point of attack. The sanitation of the area left much to be desired and, from 1917 on, the Shire Council, guided by Dr. Kay and the Commissioner of Health, waged a vigorous campaign to clean it up. This was supplemented by widespread and repeated anti-typhoid vaccination. Homes of sufferers were disinfected.

In spite of these measures, cases of fever recurred each summer in West Plane Creek. Residents of the area were shunned when they visited Mackay. Some left the district to avoid infection. An unfortunate fruit hawker, who had had the fever in 1918, was regarded for some years as a carrier although the regulation samples had been tested for typhoid and the findings were negative; cases were said to follow his visits, and he sought legal aid to clear his name. The proponents of an enteric ætiology urged the Council to intensify their sanitary efforts, but those most closely associated with the cases realized that this approach was inadequate and requested the Queensland Health Department to undertake further investigations.

Accordingly, in June, 1922, a laboratory was set up in Sarina by H. E. Brown, and during the next three months blood and fæces from practically all the inhabitants of West Plane Creek were examined for the organisms of malaria and enteric. No evidence of either disease was discovered. Soil and water samples were examined without result. Although no opportunity arose then to test new cases of fever, the failure to find a carrier dealt a further blow to the typhoid hypothesis, but the nature of the disease remained a mystery. It was popularly termed "West Plane Creek fever" or "Sarina fever", and under these names gained admission to the medical literature. Telford pointed out that the latter name was not fair to the township because there were very few cases in Sarina itself, and, as regards the former name, such cases were not confined to West Plane Creek alone. He had in mind Clarke's report in 1913 on "Mossman fever".

Since 1922, there have been few reports of fever from West Plane Creek, and no fatal case has come to our notice. A group of three cases is on record in April and May, 1941.

Since the time of the outbreak much has been learned about the fevers of North Queensland. Following the report in 1922 by Hone of a series of cases in Adelaide, South Australia, closely resembling typhus, the resemblance to typhus was recognized of certain fever cases in North Queensland also. The distinction between murine and scrub typhus was not clarified until the work of Fletcher and Lesslar (1926) in Malaya. Cases of scrub and murine typhus confirmed by *Proteus* agglutination were reported from the Cairns district by Langan and Mathew in 1935. (Reference is made by these authors to earlier, unpublished cases of Paine and Nye.) Leptospirosis in epidemic form was recognized in Ingham in 1934 (Morrissey; Cotter and Sawers), and a case of "Q" fever in the same district in 1945 (Morrissey and Derrick). Twelve cases of tick typhus were reported from the Atherton Tableland in 1946 (Andrew *et alii*). Still other entities may be present, for many cases of fever defy specific diagnosis.

A review of the Sarina story in the light of this later knowledge leads to the conclusion that "Sarina fever" was scrub typhus. The evidence may be summarized as follows:

1. The main epidemiological features of "Sarina fever"—localization to a small "island" of infection, dramatic outbreak, recurrence at the same site from time to time and later virtual disappearance, association with rain forest or its margins, incidence among men engaged in clearing, occurrence at the season when mites are most numerous—are all characteristic of scrub typhus.

2. The clinical features of "Sarina fever" that are on record are fully in accord with scrub typhus. The only notable omission is that there is no mention of an eschar.

3. Of the fevers known to occur in North Queensland, only typhoid, leptospirosis and scrub typhus could match the severity of the Sarina cases. Typhoid has already been excluded. If "Sarina fever" had been leptospirosis, jaundice, which is not mentioned, would have been an outstanding feature. In fatal cases of leptospirosis the patients are nearly always jaundiced, as well as in two or three times as many non-fatal cases. This leaves only scrub typhus. It is true that the mortality at Sarina was much higher than is usual for scrub typhus in North Queensland. The combined figures for the series reported by Mathew (1938), Heaslip (1941) and Southcott (1947) give six deaths in 195 cases, a mortality rate of 3%. In New Guinea and elsewhere, however, the incidence and mortality rate have varied considerably from one place and time to another, and severe localized outbreaks have been reported comparable to that at Sarina, as for instance on Goodenough Island, where the mortality among 51 patients was 25% (Ripley, 1946). Some of the Sarina deaths may be attributable to late hospitalization, for some patients went on working after the onset and in those days transport to the nearest hospitals at Mackay was not easy.

4. The identification of "Sarina fever" with scrub typhus is supported by the occurrence from time to time in various other places in the Mackay district, such as Habana, Mount Jukes, Midge Point and elsewhere, of cases diagnosed clinically as scrub typhus. At least two of these were fatal. A woman, aged fifty-eight years, from Habana died about 1935 with the classical symptoms including an eschar on the leg. In May, 1949, a farm worker of Mount Jukes, aged eighteen years, died about the eleventh day of illness. He had a pubic eschar, but had no serum agglutinins for *Proteus* OXK on the day of death. A recent case of scrub typhus, confirmed by *Proteus* agglutination, will be presently described.

5. "Sarina fever" has not established itself as a clinical (apart from being a geographical) entity. Although it was declared a notifiable disease in 1934 in order to obtain precise information about its incidence, no case has since been notified under that name either from the Sarina district or elsewhere in Queensland. Possible cases in recent years would be likely to be regarded and notified as scrub typhus.

It is not suggested that every case of fever at West Plane Creek would have been scrub typhus. Various infections that visit other places could visit there also. Some of the cases may perhaps have been tick typhus, for the entry of men into new "scrub" areas to open them up for cultivation would expose them to tick typhus as well as to scrub typhus if the infected vectors were present. Tick typhus, which is present in north and south Queensland as a mild disease, has not so far been recorded from the Mackay district.

Clinical Record.

We pass now from historical considerations to an account of certain patients with fever who have been treated in the Mackay District Hospital during the last three years.

It may be mentioned that there is a high incidence in Mackay of urinary infection, which may be symptomless or accompanied by fever. An early step in the investigation of a fever patient is microscopic examination of the urine.

For specific diagnosis, sera are submitted to the Laboratory of Microbiology and Pathology, Brisbane, where they are tested for agglutination with various organisms. During most of the period under review, the test organisms used routinely were: *Proteus* OX19, *Proteus* OXK, *Coxiella burneti*, *Leptospira icterohæmorrhagiæ*, *L. canicola*, *L. australis A*, *L. australis B*, *L. pomona*, *L. mitis*, *Salmonella typhosa*, *S. paratyphi*, *S. schottmülleri* and *Brucella abortus*. In the cases here reported, no significant agglutination was found with organisms other than those mentioned in the case notes. In addition, complement fixation tests were carried out in some cases with *Coxiella burneti* and other rickettsial antigens.

Scrub Typhus.

In this case of scrub typhus, the outstanding clinical features were headache, adenitis, cough, hypotension and conjunctival injection. There was no eschar or rash.

CASE I.—J.S., aged twenty-nine years, a canecutter of Silent Grove, was admitted to hospital on July 4, 1950. This was the eighth day of his illness, which had begun with headache, fever, joint pains, cough and slight abdominal pain. He had slept poorly all the week and had developed a pain which he described as "nagging" at the right costal margin.

On admission to hospital the patient looked ill. The temperature was 102° F., the pulse rate 120 per minute, the respiratory rate 30 per minute, and the blood pressure 95 millimetres of mercury, systolic, and 55 millimetres, diastolic. The tongue was furred and the throat mildly inflamed. There was some tenderness in the epigastrium and right hypochondrium. The urine contained no chemical abnormality. There was slightly diminished expansion of the left side of the chest. No eschar or rash was found.

A provisional diagnosis of early left lobar pneumonia was made, and penicillin treatment was started—an initial dose of 100,000 units was followed by 30,000 units three-hourly until July 9, when the suspicion of leptospirosis caused the three-hourly dose to be increased to 100,000 units. Penicillin administration was ceased on July 19.

The cough was unproductive and persisted for some days. No adventitia developed in the lungs, and an X-ray film

taken on July 5 revealed only a doubtful slight infiltration of the right lung base. Another film taken on July 14 showed no change. On the eleventh day palpable glands in axillæ and groins and conjunctival injection were noted. On the same day the white cell count was 12,000 per cubic millimetre with 84% of neutrophile cells. The fever lasted nineteen days (see Figure III). The pulse rate was relatively slow throughout the illness, the highest rate recorded after that on admission being 108 per minute.

The blood pressure varied somewhat but remained below normal until the fever had subsided. The urine was tested daily. On one day only, the twelfth, albumin was present. Microscopic examination on the eleventh day showed a few leucocytes and red cells.

The headache was persistent and troublesome. On one occasion it was severe enough to require morphine for its relief. There was no stiffness of the neck. The patient complained of pain in the joints and the back throughout the

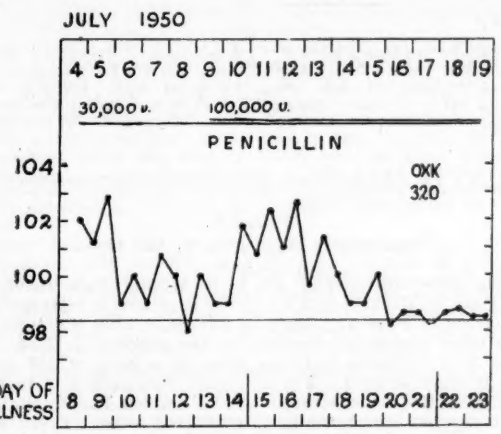

FIGURE III.
Temperature chart, Case I, scrub typhus.

fever. The deep reflexes were noted to be much depressed on the fourteenth day. Vomiting was not troublesome, and anorexia was not severe. The patient was not delirious at any stage and was not drowsy or irritable.

On July 14, though still febrile, he felt much better, and this improvement was maintained. He was discharged from hospital on August 1. When he was examined again a week later he had no complaints except slight weakness.

Serum agglutination with *Proteus* OXK to a titre of 1:640 on the twenty-first day of illness established the diagnosis of scrub typhus. It is likely that he acquired his infection while shooting in mountains covered with tropical jungle near the farm on which he worked.

Murine Typhus.

Murine typhus is represented by two cases. Both patients lived in the city; they were the only city dwellers in this series. Contact with rats was elicited with both.

CASE II.—D.D., aged fifteen years, was working for a large Mackay grocery firm in the produce section where rats and mice were prevalent. He first felt ill on Thursday, March 9, 1950, went fishing for the week-end and was admitted to hospital on March 13. He complained of shivering attacks, dizziness on standing, anorexia and a foul taste in his mouth. He said that his head felt heavy but he had no definite headache.

On his admission to hospital the temperature was 103° F., the pulse rate 74 per minute, the respiratory rate 20 per minute and the blood pressure 90 millimetres of mercury, systolic, and 55 millimetres, diastolic. The tongue was dry and dirty and the pharynx slightly injected. The lungs, heart and abdomen were clinically normal. The lymph glands were palpable in the left groin and in both epitrochlear regions. The urine was chemically normal but contained a few pus cells on microscopic examination. The white cell count (March 16) was 5300 per cubic millimetre with 64% neutrophile cells.

Sulphadiazine, one tablet four-hourly, was administered for four days and then penicillin 100,000 units eight-hourly for four days. The temperature gradually subsided and remained normal on and after March 26 (see Figure IV). The pulse rate did not rise above 98 per minute throughout the illness. The urine remained chemically normal and was microscopically clear when examined on March 22. The blood pressure was recorded as 100 millimetres of mercury, systolic, and 50 millimetres, diastolic, on March 23.

The patient did not complain of headache. He took food and fluids willingly and generally gave the impression that he was less ill than his temperature chart would indicate. He was discharged from hospital on March 30 and was examined again on April 14, when he felt well but said that his legs had been very weak for a few days after his discharge from hospital.

Serum taken for agglutination on March 28 gave a titre of 1:320 with *Proteus* OX19.

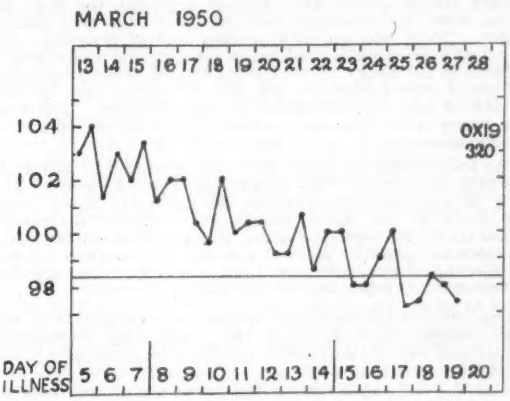

FIGURE IV.
Temperature chart, Case II, murine typhus.

CASE III.—E.P., aged forty-five years, a printer, of Mackay, was admitted to hospital on March 27, 1951. This was the ninth day of his illness. He had complained of fever, nausea and anorexia, frontal headache and pain in the back of the neck. He had had several rigors and for the past few days an unproductive cough. He had had slight dysuria but no frequency of micturition and no diarrhœa. Before admission to hospital he had been given 22 capsules of "Chloromycetin" (two capsules at six-hourly intervals) with no appreciable effect.

On his admission to hospital the temperature was 103° F., the pulse rate 120 per minute, the respiratory rate 24 per minute and the blood pressure 120 millimetres of mercury, systolic, and 60 millimetres, diastolic. There was no stiffness of the neck. The heart and lungs were clinically normal, and an X-ray examination on March 28 showed normal lung fields. The urine was chemically clear but microscopically showed a moderate number of pus cells and an occasional red blood cell. The white cell count was 6800 per cubic millimetre, with 80% of neutrophile cells.

The only treatment given was symptomatic treatment and a course of sulphadiazine (two tablets four-hourly) from March 27 until April 4. The temperature fell by lysis, becoming finally normal on April 4. The pulse rate ranged from 120 to 72 per minute. Severe headache persisted throughout the febrile stage, insomnia was troublesome, and *herpes labialis* developed. The blood pressure did not vary significantly. There was no rash or adenitis, and no adventitia developed in the lungs. The urine was microscopically clear on April 4. The patient was discharged from hospital on April 7.

Proteus OX19 was agglutinated to titres of 1:80 and 1:160 with serum taken on April 9 and June 1.

The patient denied association with rats or ticks. However, inquiry disclosed that rats were often seen inside the premises where he worked, and, for this, proximity to three cafes were blamed.

"Q" Fever.

There were three cases diagnosed as "Q" fever. A short course of eight days of fever in a young man contrasts with a duration of twenty-nine days in an elderly man.

CASE IV.—P.L., aged twenty-three years, a bulldozer driver of Mirani, was admitted to hospital on August 24, 1951, at 7 p.m. This was the fourth day of his illness. During the previous month he had worked at Owen's Creek for two weeks, then at Eton quarry for one week and lastly on the river bed at Mirani for one week.

He had had shifting pains in the fingers, elbows and knees and some pain and stiffness in the muscles of his back and neck. He felt worse in the afternoons than in the mornings. He had had two or three rigors and an intermittent frontal headache. He had continued working for three days, but on the fourth day his shoulders were very stiff and painful, and he was sweating freely, had developed an unproductive cough and felt generally worse.

On his admission to hospital his temperature was 99° F., his pulse rate 100 per minute and his respiratory rate 24 per minute. His blood pressure was 130 millimetres of mercury, systolic, and 80 millimetres, diastolic. He did not look very ill, he was not drowsy, and his headache was not severe. The axillary lymph glands were slightly enlarged, the liver and spleen were not enlarged, but the liver edge was slightly tender. There was no rash or conjunctival injection. The deep and superficial reflexes were normal, and there was no stiffness of the neck. No muscle tenderness could be detected, the knees were tender but not hot or swollen, and he complained of pain on movement of the shoulders. The heart and lungs were clinically normal. The urine showed no abnormalities.

The only treatment given was administration of salicylates and general nursing. The temperature ranged from 99° F. to 101·6° F. and reached normal on the ninth day of the illness. The pains in the joints, cough and headache disappeared before then. He was discharged from hospital on September 1. He was examined again on September 7, when he complained of slight morning headaches; but on his next attendance on September 17 he was quite well.

Tests of sera taken on September 7 and September 17 gave agglutination titres of 1:80 and 1:160, and complement-fixation titres of 1:64 and 1:128 with *Coxiella burneti* antigens.

CASE V.—A.B., aged sixty-seven years, a fruit and vegetable farmer of Seaforth, became ill on June 5, 1952, with severe frontal headache and slight epigastric pain. Next day he developed pain in the lower part of his chest on the left, which was worse on coughing. He had had a slight cough for years. He felt very tired.

On admission to hospital on June 7 he appeared rather sick. No abnormal signs were found in the alimentary, respiratory or nervous systems, but hypertension (210 millimetres of mercury, systolic, and 105 millimetres, diastolic) was discovered. The fever ran a rather irregular course (see Figure V) and lasted altogether twenty-nine days. The pulse rate was comparatively slow at first, but tended to rise during the latter half of the illness. He complained from time to time of pain in the chest. X-ray films of the chest on June 21 and July 3 showed no abnormality. On July 3 a few rhonchi were heard on the right side. On June 14 some pink macules appeared on the abdomen and persisted for a week. The leucocyte count was 5500 per cubic millimetre on June 9, and 11,000, with 52% neutrophile cells and 48% lymphocytes, on June 23.

The course was not apparently influenced by penicillin, sulphamezathine or chloramphenicol, though it may be noted that the course of chloramphenicol was restricted through a misunderstanding about supplies. A dose of 0·75 gramme was given six-hourly for four days, followed by 0·25 gramme six-hourly for two days (total 14·25 grammes).

Serum collected on June 11 failed to agglutinate *Coxiella burneti*; that collected on June 24 agglutinated it to a titre of 1:40.

CASE VI.—W.D., a dairy farmer, aged fifty-six years, of Yakapari, was twice admitted to hospital with febrile attacks. The first began on March 2, 1951, the fever lasting eleven days, the second on March 31, when there were seven days of fever. His serum on April 27 agglutinated *Coxiella burneti* at a titre of 1:30—unfortunately tests with leptospiræ were not carried out on that occasion. Five months later agglutinins for *Leptospira pomona* were found (titre 1:300), but those for *C. burneti* had disappeared. Complement-fixing antibodies for *C. burneti* were, however, present, at a titre of 1:32. It is impossible now to interpret the test results with certainty in relation to the febrile episodes, particularly as the first fever was accompanied by pyuria, but the presence of antibodies to both *C. burneti* and *L. pomona* confirms the presence of these organisms in the district.

Leptospirosis.

Leptospirosis is comparatively common. Six cases have been recognized, and in all the infecting type was *Leptospira pomona*. Some facts about these cases are set out in Table I, including the serological results on which the diagnosis was made. There were two examples of two cases in a family. In all cases there was direct contact with cattle or pigs. Five of the cases occurred during the wet season.

The localities mentioned in Table I show that *L. pomona* is widely distributed throughout the Mackay district. This is confirmed by veterinary experience.

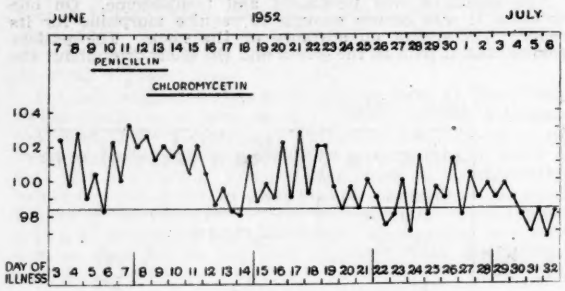

FIGURE V.
Temperature chart, Case V, "Q" fever.

We were informed by Mr. L. G. Walker, then Inspector of Stock at Mackay, that an outbreak of bovine leptospirosis occurred in February, 1950, on a farm at Blue Mountain, in the same locality as the farm of the patients in Cases VII and VIII. Twelve lactating cows in a herd of 103 dairy cattle were affected. The diagnosis was confirmed in four cases by the demonstration at the Animal Health Station, Brisbane, of leptospiræ in formalinized urine. There were suspected cases of leptospirosis among the cattle on two adjacent farms and another proved outbreak in February, 1950, on a farm on the Connors Range near Sarina. It is likely that these bovine infections were due to *L. pomona*. Three of the farmers on these farms suffered from a severe febrile illness at about the same time that their cattle were sick. More recently bovine leptospirosis has appeared on a farm at Eungella.

Leptospiroses of the types seen in the far northern canefields have not been recognized in the Mackay district. However, McDougall (1944) has reported that *Rattus conatus* is present, the Mackay district being the southern limit of its known range. This rat (at the time incorrectly identified as *Rattus culmorum*) was found by Sawers (1938) to be the main reservoir of leptospiral infection in the canefields around Ingham.

The cases of leptospirosis in our series had a sudden onset; three of the patients were already in hospital by the second day. Severe headache and muscular pains were prominent symptoms. In two cases the presence of meningeal symptoms led to the performance of lumbar puncture. The conjunctivæ were injected in three cases. The urine on microscopic examination usually showed some pus and red cells. Other features present were a rash (three cases), lymphadenitis (two cases), leucocytosis (one case). No patients had jaundice. The duration of the fever in five cases was seven, seven, seven, eight and nine days. The pulse rate was rather slow in comparison with the temperature. Three patients were treated with penicillin, without any apparent benefit.

Clinical accounts of the cases follow.

CASE VII.—C.J.K., aged twenty years, a dairy farmer of Blue Mountain, was admitted to hospital on January 2, 1951, which was the fifth day of his illness. He had already had a course of some "sulpha" drug, which he had finished twenty-four hours previously. He had been feverish from the onset, and the day before admission he developed pain in both sides of the chest on deep inspiration and a slight cough with blood-stained sputum.

TABLE I.

Cases of Leptospirosis Pomona.

Case Number.	Sex.	Age.	Residence.	Occupation and Comment.	Date of Onset.	Serum Agglutination.	
						Day of Illness.	Reciprocal of Titre for *L. pomona*.
VII	M.	20	Blue Mountain.	Dairy farm worker; keeps pigs; goes barefoot.	29/12/50	18	300
VIII	M.	14	Blue Mountain.	Dairy farm worker; brother of previous patient and works with him.	1/1/51	15	300
IX	M.	19	Farleigh.	Canefarmer; five cattle on farm but no pigs; duck shooting at weekends.	9/2/51	20	100
X	M.	23	Orkabie.	Canecutter; two house cows.	6/8/51	9	0
						26	100
XI	M.	18	Netherdale.	Mixed farmer; fruit, vegetables, cattle, 100 pigs; goes barefoot.	27/2/52	3	0
						20	300
XII	F.	7	Netherdale.	Schoolgirl; lives on same farm as previous patient, her brother.	3/3/52	24	300

On admission to hospital he had a temperature of 100° F., a pulse rate of 102 per minute, and a respiratory rate of 26 per minute. His blood pressure was 95 millimetres of mercury, systolic, and 65 millimetres, diastolic. He looked pale and ill. Conjunctival injection was not noticeable. There were a few raised pink marks on his legs. The air entry at both lung bases was slightly diminished, but there were no adventitia. X-ray examination of the lungs showed only slightly increased basal markings. No chemical abnormalities were detected in the urine; microscopic examination was not made.

The patient was regarded as suffering from early pneumonia, and penicillin was given for six days in doses of 60,000 units three-hourly. The temperature ranged from 99·8° F. to 102° F. and reached normal on the eighth day of the illness. The maximum pulse rate was 114 per minute. The blood pressure was 125 millimetres of mercury, systolic, and 55 millimetres, diastolic, on the sixth day.

On the same day a pink maculo-papular rash was evident on chest, abdomen and legs. This faded on the eighth day and may have been due to the "sulpha" drug. The cough and headache persisted until the temperature was normal. The lungs were clinically normal on the eighth day. White cell counts showed 18,000 per cubic millimetre on the day of admission to hospital and 16,000, with 75% neutrophile cells, on January 8.

The patient was discharged from hospital on January 15 and was examined again on February 9, when he felt quite well and had returned to work.

CASE VIII.—G.K., aged fourteen years, became ill three days after his brother, C.J.K., with whom he had been working. The onset was sudden, with fever, severe headache, muscular pains and anorexia. He was delirious for a few hours on the second evening. On the third day central abdominal pain was added to the other symptoms. On the fourth morning he vomited dark material, which, from his description, was thought to be altered blood. He had loose stools during the first three days and also had an unproductive cough.

On his admission to hospital on the fourth day the temperature was 101° F., the pulse rate 116 per minute and the respiratory rate 24 per minute. He had a dry cough, but no physical or X-ray abnormalities were detected in his lungs. The tongue was clean and moist. There was some central abdominal tenderness, the liver edge was tender, and the spleen was not palpable.

No antibiotics were administered. The temperature ranged around 100° F. to 101° F., reached a maximum of 103° F. early on the seventh day and fell to normal on the eighth day. The highest pulse rate recorded was 100 per minute. Headache persisted throughout the fever, and there was slight sacral backache also. There was no vomiting or diarrhœa after admission to hospital. The abdominal pain was not severe and persisted for only two days.

On the fifth day, red, slightly tender lumps appeared on the left elbow and left shin. They resembled *erythema nodosum*. They had disappeared by the eighth day. There was no dysuria, and no albumin or other chemical abnormality appeared in the urine. Microscopically, the centrifuged urine on January 5, 1951, showed a moderate number of pus cells and red cells; it was free from them on January 9. On January 5 the white blood cells numbered 6000 per cubic millimetre, with 80% neutrophile cells.

The patient was discharged from hospital on January 15, but was seen several times afterwards, when he complained of headache and lassitude. This was in contrast to his older brother, who rapidly regained normal health. However, G.K. was as tall and well developed as a youth of eighteen or nineteen years and had returned early to heavy work more suitable for a grown man. He was last seen on February 23, when he was improving.

CASE IX.—P.V., aged nineteen years, a sugarcane farmer of Farleigh, was admitted to hospital on February 10, 1951, which was the second day of his illness. The first symptom had been severe headache, followed quickly by stiffness of the neck, vomiting, abdominal discomfort and slight dysuria.

On admission to hospital he had a temperature of 100° F. and a pulse rate of 104 per minute. There was moderate stiffness of the neck, and the knee jerks could not be elicited, but the other deep and the superficial reflexes were normal. There were no abnormal physical signs in the lungs or heart. The liver and spleen were not enlarged, and there was no abdominal tenderness. No chemical abnormalities were detected in the urine then or subsequently, but microscopic examination on February 13 showed a moderate number of pus cells and a few red blood cells.

Lumbar puncture was performed. The cerebro-spinal fluid was normal and not under increased pressure.

The temperature ranged from 99·4° F. to 103° F. and fell to normal on February 18, which was the tenth day of the illness. The maximum pulse rate was 110 per minute, and the blood pressure remained within normal limits. The patient complained of severe headache throughout the febrile period.

Palpable lymph glands in axillæ and groins were noted on the third day and also slight tenderness of the liver edge, but no palpable enlargement of the liver or spleen developed. On the fifth day the white cell count was 7000 per cubic millimetre, with 82% neutrophile cells. On the sixth day the deep and superficial reflexes were all normal, but the neck stiffness persisted until the eighth day. Conjunctival injection was present and persisted throughout the fever, being very noticeable on the eighth day.

A fine macular rash appeared on the trunk and limbs on the sixth day. It faded in a few days and may have been due to the sulphadiazine which was administered from admission to hospital until the sixth day. No physical signs developed in the lungs. The patient complained of abdominal discomfort and a sense of fullness, but there was no distension. He was not constipated and there was only occasional vomiting.

Penicillin, 100,000 units three-hourly, was administered during the last few days of the illness (February 16 to February 20). That and the sulphadiazine were the only treatment given other than analgesics for the headache.

The patient was discharged from hospital on February 20. He was well when last seen on February 28.

CASE X.—E.D.C., aged twenty-three years, a canecutter of Orkabie, 54 miles south of Mackay, was admitted to hospital on August 7, 1951, on the second day of illness. Features of the illness were fever (up to 103° F.), generalized muscular pains especially severe in the back, severe frontal headache, marked conjunctival injection and moderate enlargement of axillary and inguinal glands. There was no rash, stiffness of the neck, enlargement of liver or spleen, significant variation in the blood pressure or increase in leucocytes. He was treated with penicillin, 100,000 units six-hourly, from admission to hospital until August 11. The last day of fever was August 12.

He was discharged from hospital on August 14, still rather weak, but reported on August 31 that he was very well.

He stated that for the month before his illness he was fully engaged in cutting cane and had no contact with stock except for two house cows which were in good health.

CASE XI.—H.B., aged eighteen years, a farm worker of Netherdale, was admitted to hospital on February 28, 1952. He had been working on a mixed farm, where the presence of 100 pigs indicates the likely source of his infection.

The day before admission to hospital he had noticed first that his eyes were painful and the light hurt them; then he developed headache and backache. The next day these symptoms were more severe, and his legs were painful when he attempted to walk.

On his admission to hospital his temperature was 102·6° F., his pulse rate was 100 per minute, his respiratory rate 24 per minute and his blood pressure 125 millimetres of

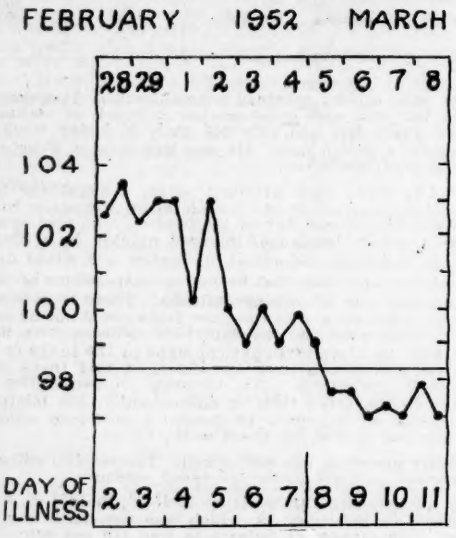

FIGURE VI.
Temperature chart, Case XI, Pomona leptospirosis.

mercury, systolic, and 80 millimetres, diastolic. He was complaining of severe headache, backache and pain in the elbows and knees. The conjunctivæ were injected, and there was marked photophobia. The tongue was coated and the pharynx normal. Nothing abnormal was detected in the heart, lungs and abdomen. The deep and superficial reflexes were normal. There was some stiffness of the neck. No chemical abnormalities were detected in the urine.

That night he had a mild rigor. The next day the conjunctival injection and the neck stiffness had increased, and the sterno-mastoid muscles were tender to palpation; otherwise his condition was unchanged. Lumbar puncture was performed. The cerebro-spinal fluid was clear and under a pressure of 145 millimetres of water. It contained five cells per cubic millimetre and 25 milligrammes of protein per 100 mils. No organisms were seen in a film stained by the Gram method.

The only treatment given was a short course of sulphadiazine and salicylates. The temperature remained high until the morning of the fifth day and then rapidly subsided. There were seven days of fever (Figure VI). The pulse rate did not rise above 108 per minute, and there was no significant variation in the blood pressure. The headache and muscular pains remained severe for several days, the headache not finally disappearing until the tenth day. No rash or adenitis developed.

The leucocytes numbered 11,000 per cubic millimetre on February 29, with 90% neutrophile cells. Microscopic examination of the urine on the same day showed numerous leucocytes and red cells. These had disappeared on March 6. The urine remained chemically normal.

He was discharged from hospital on March 10.

CASE XII.—G.B., aged seven years, sister of the previous patient and residing with him, had a mild illness beginning on March 3, 1952. The fever was irregular and recurred after an afebrile period of seven days; the temperature finally became normal on March 26. There was no albuminuria, glandular enlargement, jaundice or rash. Some pus in the urine at the beginning of the illness raised a suspicion of pyelitis; the urine later became free of pus.

Fever of Undetermined Origin.

Cases of fever present themselves from time to time, in which the findings of serological tests are negative. Most of them are fairly mild. An account is given below of a more severe case in which the fever lasted fifteen days. It closely resembled scrub typhus clinically, but the serum failed to agglutinate *Proteus* OXK or the other test organisms. In these respects it is like cases met with not infrequently in the far north of Queensland.

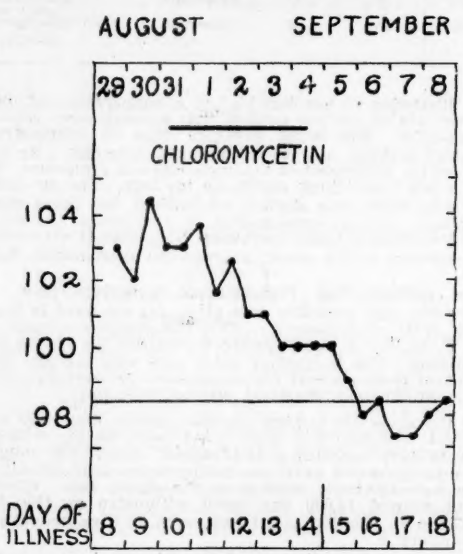

FIGURE VII.
Temperature chart, Case XIII, fever of undiagnosed cause.

CASE XIII.—J.G.R., aged twenty-three years, a sugarmill worker of Farleigh, was admitted to hospital on August 29, 1951. This was the eighth day since the onset of symptoms. He stated that he had been shooting near The Leap about twenty-seven days before he first felt ill. The first symptoms had been pain in the back and chest, which passed off leaving him tired and depressed but able to continue work and to go shooting again. Two days prior to admission to hospital he felt worse and went to bed with a shivering attack. On the next day he noticed a rash on the back of his hands which spread rapidly all over his body. He had had a headache intermittently all the week and a cough but no coryza. He also complained of stiff painful joints.

On admission to hospital he was pale and shivering with a temperature of 103° F., a pulse rate of 100 per minute and a blood pressure of 130 millimetres of mercury, systolic, and 90 millimetres, diastolic. There was a red maculo-papular rash which faded on pressure on the limbs and trunk. There was no eschar. The inguinal and axillary lymph glands were palpable and tender. There was slight generalized abdominal tenderness. The liver and spleen were not enlarged. The heart and lungs were clinically normal, the tongue was clean, the pharynx was slightly injected; the conjunctivæ were slightly injected. There was no neck stiffness, and the deep and superficial reflexes were normal. The patient remained feverish until the evening of September 5, when the temperature had fallen to normal by lysis (see Figure VII). The pulse rate did not rise above 110 per minute, and there was no significant change in the blood pressure. The rash began to fade on September 3. It was followed by a fine desquamation of the hands and feet. The headache, which was retroorbital in character and moderately severe, persisted until September 4. The patient exhibited some photophobia and seemed to be slightly deaf during the febrile period. The deep reflexes were depressed for a few days

during the later part of the week after admission to hospital. He was drowsy until his temperature became normal, and on September 2 he showed definite mental confusion. The cough persisted for about a week, but the lungs remained clinically normal. The urine was tested daily and showed no abnormalities.

Penicillin was administered, 400,000 units eight-hourly, from his admission to hospital until September 7 without any detectable effect. Chloramphenicol was given from the morning of August 31 until September 4 in doses of 250 milligrammes four-hourly for forty-eight hours and then in 500 milligramme doses four-hourly. It produced no obvious decrease in the symptoms, although the temperature gradually declined during its use.

The patient was discharged from hospital on September 20 and was seen several times subsequently when he reported to have further blood samples taken. He was quite well.

Three samples of serum, collected on August 29, September 17 and September 28, were examined for agglutination with the thirteen test organisms and with five new North Queensland types of *Leptospira*, but none was found. The second and third samples were also tested for rickettsial complement fixation. None was found with murine typhus and "Q" fever antigens, but with rickettsialpox antigen the second serum gave a titre of 1:8 and the third 1:16. Our use of rickettsialpox antigen for the diagnosis of cases of Queensland tick typhus is still experimental, and the significance of fixation at titres of 1:8 and 1:16 is not clear.

Discussion.

The selected cases here described show that the diagnosis of a case of fever in the Mackay district requires consideration of at least three rickettsioses—scrub typhus, murine typhus and "Q" fever—and at least one type of leptospirosis. Future work may be expected to extend the list.

Although the present report is far from being a complete record, it is evident that the relative incidence of fever at Mackay is much less than in the high-rainfall area from Ingham to Mossman, further north in Queensland. Whereas these 13 cases have been collected at the Mackay Hospital over a period of nearly three years, the Innisfail Hospital, for instance, with half the number of beds would be able to collect a comparable series in well under twelve months.

Another important difference relates to the profusion of leptospiral types in the northern area, where unpublished work shows that at least eleven types cause human infection. Leptospirosis as an occupational disease of canefield workers is, so far, unknown in Mackay. These differences in incidence and type of fever are associated with a greater temperature and a much greater humidity further north.

In a comparison with southern Queensland, the essential difference is the presence of scrub typhus in the Mackay district.

Summary.

The Mackay district is for several reasons important in the study of the endemiology of certain febrile diseases. As a high rainfall area, circumscribed by dry belts, it comprises a distinct climatic subregion.

In this district in 1915 to 1922 there occurred a severe outbreak of fever, which was reported under the name of "Sarina fever". A review of the outbreak leads to the conclusion that "Sarina fever" was scrub typhus.

This conclusion is supported by the occurrence from time to time of cases of scrub typhus in various places in the Mackay district. One recent case is described.

With the acceptance of "Sarina fever" as scrub typhus, the head of Plane Creek (latitude 21° 30′ south) near Sarina becomes the most southerly place in the world from which scrub typhus has been recorded.

Six cases of leptospirosis of pomona type are reported. All the patients were associated with cattle or pigs. There have also been outbreaks of leptospirosis among cattle.

Cases of murine typhus and "Q" fever are also reported, as well as a case of fever in which we have been unable to reach a diagnosis.

The fever pattern in the Mackay district includes at least three rickettsioses—scrub typhus, murine typhus and "Q" fever—and leptospirosis. It differs from that of south Queensland by the presence of scrub typhus, and from that of the far north of Queensland by a much lower incidence of fever cases and by a comparative sparsity of leptospiral types.

Acknowledgements.

We are greatly indebted to the practitioners of Mackay, in particular Dr. C. E. Williams, Dr. M. J. Gallagher, Dr. R. Grant, Dr. P. W. Hopkins, Dr. S. C. Williams, Dr. R. Courtice and Dr. E. F. Reye, for clinical information, also to Dr. D. Gordon, of Brisbane. Mr. L. A. Payne, Shire Clerk of Sarina Shire, and Mr. J. Williams, Health Inspector of Pioneer Shire, went to much trouble in searching old records. Councillor J. P. Jackson, Chairman of Sarina Shire, Mr. J. C. Nicholson, who was chairman at the time of "Sarina fever", other residents of West Plane Creek, Mr. J. Seabrook, Health Inspector of Sarina Shire, Mr. I. Murray, Town Clerk, Mackay, Mr. L. G. Walker, Stock Inspector, Mr. R. A. Burke, State Health Inspector, and Mr. John H. Williams, junior, and others provided valuable information and assistance. Mr. G. E. Mortimer carried out pathological investigations on the patients at Mackay Hospital, and Mr. D. J. W. Smith assisted in the serological tests in Brisbane. Dr. J. Legg kindly furnished details of the leptospiral tests of cattle, and Mr. I. Ferguson of sugar statistics.

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Addendum.

Another case of scrub typhus has recently occurred. The patient, B.M., aged thirty-nine years, was admitted to the Mackay District Hospital on March 22, 1953, on the sixth day of illness. He was a canefarm labourer from Balnagowan (ten miles west of Mackay) and had been cutting dead guinea grass. The illness was mild, the main complaint being a troublesome headache. There was no rash or eschar. The fever lasted in all twelve days. The agglutination titres with *Proteus* OXK were 0 on the ninth day, 1:640 on the twenty-sixth day and 1:80 on the sixty-eighth day.

THE SURGICAL TREATMENT OF SPONTANEOUS SUBARACHNOID HÆMORRHAGE.

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Historical Survey.

THE occurrence of aneurysms on intracranial arteries has long been recognized, the earliest account being attributed to Biumi of Milan (1765). A case of ruptured aneurysm of the basilar artery was reported by Blackall (1816), and sporadic reports appeared in the ensuing years until Beadles (1907) was able to collect accounts of 555 cases. Symonds (1923) stimulated further interest in a paper which conclusively related spontaneous subarachnoid hæmorrhage with the presence of an aneurysm and pressed for the clinical recognition of these lesions. Moniz (1934), who introduced cerebral angiography as a clinical procedure, supplied an unparalleled means of investigation which enabled the exact location and origin of aneurysms to be determined by outlining them with radio-opaque dye injected into the carotid artery. Moreover, angiomatous vascular malformations of the brain, which account for a number of subarachnoid hæmorrhages, can be similarly revealed, and the impetus to the surgical treatment of both types of lesion during the past two decades stems largely from this work. Before its advent, only those aneurysms sufficiently large to produce localizing signs could be suspected, and often their aneurysmal nature did not become evident until the mass had been exposed at operation. Again, the adequacy of the anastomotic circulation can be proved angiographically—a point of great value should it be necessary to ligate one or other of the carotid arteries.

Originally, cerebral aneurysms were not uncommonly found in association with infective endocarditis (the mycotic aneurysms), arterial degenerations and syphilis, whilst a congenital origin was attributed to some. During this century, it has become increasingly clear that syphilis is now but rarely a predisposing factor, and more recently, since severe infections have been so successfully combated with antibiotic drugs, embolic aneurysms and ruptures are seldom seen. Today, they are often associated with arteriosclerosis or have a congenital source of development, although the exact nature of the basic congenital fault in the artery which permits the eventual aneurysmal "blow-out" is not the subject of universal agreement. Forbus (1930) alleged that gaps in the medial musculature at points of bifurcation were the weak spots; but Glyn (1940) challenges the validity of this view and considers that the elastic tissue concentrated in the cerebral arteries is particularly susceptible to injuries and degenerations. Whatever the cause may be, it must be borne in mind that congenital aneurysms may be associated with variations from the normal configuration of the circle of Willis, and with congenital arterial defects elsewhere in the body, as for example aortic coarctation.

It is with the present-day surgical approach to the problems of intracranial aneurysms and spontaneous subarachnoid hæmorrhage that this paper is mainly concerned.

Clinical Features of Intracranial Aneurysms.

Intracranial aneurysms have often come to light unexpectedly on the cerebral vessels of patients who have died from other unrelated causes, and in consequence it is widely accepted that they may exist during life for long periods without producing any deleterious effects. On the other hand, they frequently announce their presence in dangerous fashion by causing spontaneous subarachnoid hæmorrhage in patients who have seemingly been hitherto in good health. Between these two extremes, there are many heralded in less dramatically by the sudden appearance of isolated or multiple cranial nerve palsies, or by other symptoms such as chronic recurring headache. This type of onset should be regarded as a timely warning of impending subarachnoid hæmorrhage and treatment promptly instituted.

The most striking event in the histories of the vast majority of patients who are capable of giving one after having had a subarachnoid hæmorrhage is the dramatic onset of an intense headache, which may be strictly localized or completely generalized, and which is, perhaps, most often experienced first in the occipital and nuchal regions, though the site of the headache does not give any reliable clue to the position of the bleeding vessel. After the headache may be expected interference with consciousness, which may vary from slight clouding to deep coma from which the patient cannot be roused. Sometimes a few epileptiform fits occur, whilst the most constant physical finding is neck rigidity not infrequently accompanied by bilateral Kernig and Babinski signs. These violent events point to the diagnosis, which should be confirmed by lumbar puncture, the presence of uniformly blood-stained cerebro-spinal fluid making the diagnosis absolute.

A short digression may not be out of place at this juncture. The withdrawal of large quantities of cerebro-spinal fluid in these early stages is to be deprecated for fear that such a measure should merely make room for further bleeding. It is possible that a balance of pressure between the cerebro-spinal fluid and the leaking blood has been achieved so that natural arrest has been able to occur, and diminishing the cerebro-spinal fluid pressure might allow the sealing clot to be disturbed with resultant fresh hæmorrhage. For this reason, no more than a few cubic centimetres of fluid should be removed for diagnostic purposes in the earliest stages; in later days, when a patient is recovering and has intractable headache, the removal of blood-stained fluid often affords enormous relief.

Once the diagnosis is certain, the subsequent course depends upon the size of the rupture, the cessation, continuation or recurrence of bleeding and the extent of destruction of nervous tissue in the neighbourhood. Recovery, when it occurs, may be complete, or there may be an array of neurological sequelæ, the nature of which will depend upon the physiological function of the region damaged. Cranial nerve palsies are prone to appear and have value as localizing signs; but in the majority of cases of subarachnoid hæmorrhage, there is an absence of neurological signs to indicate on which side the bleeding is taking place, so that one must usually rely on angiography to provide this vital information (*vide infra*).

Cranial Nerve Palsies.

When paralyses of cranial nerves occur as the primary sign, they should be regarded as a warning of impending subarachnoid hæmorrhage and treated promptly. These palsies are probably caused by weakening of the sac wall, which allows its rapid expansion; there is also undoubtedly, in a number of cases, local leakage of blood, the resulting thrombotic mass then adhering to neighbouring nerves and interfering with their physiological function. This factor of expansion is a significant one, because it is the prelude to rupture—and no one can say just how long a time will elapse before it may occur.

Intracranial aneurysms, according to their location, may affect any cranial nerve; but some nerves are far more likely to be injured than others. Thus, the second, third, fifth and sixth pairs are particularly susceptible when the aneurysm lies along the carotid system, and the third to twelfth nerves may be affected by basilar and vertebral sacs.

Aneurysms on the anterior cerebral and anterior communicating arteries lie toward the mid-line and so are particularly prone to cause injury to the optic nerves and chiasm, with the production of a variety of visual field defects. Bitemporal hemianopia is not uncommon, as also is a central scotoma in one eye with a temporal quadrant defect in the other. If the sac rests mainly on one optic nerve, unilateral blindness may result. Rarely does a

posteriorly directed aneurysm lie against the optic tract to cause homonymous hemianopia.

If subarachnoid hæmorrhage has already occurred, changes may appear in the fundi; papilloedema or subhyaloid hæmorrhages may be discovered, the latter being pathognomonic of the condition. On the other hand, if there has been prolonged pressure on the optic nerves, a degree of optic atrophy should not be surprising.

An isolated oculomotor nerve palsy is frequently the outstanding feature of aneurysms arising from the supraclinoid portion of the internal carotid or the posterior communicating arteries. These sacs tend to be directed posteriorly and hang down upon the nerve before it enters the cavernous sinus. Paralysis may be partial or complete and is characterized by ptosis, and by loss of upward, downward and medial movement of the eye with a dilated fixed pupil.

Abducent nerve palsies are not common with aneurysms on the supraclinoid portion of the internal carotid, but are almost to be expected when the aneurysms lie in the cavernous sinus; however, as the latter are not a source of subarachnoid hæmorrhage, they are irrelevant to the present discussion.

The trigeminal nerve is subject to irritation, especially by rapidly expanding aneurysms in the supraclinoid position. Intense pain in the frontal region above the eye on the same side as the lesion is an almost invariable complaint under these conditions. However, this is also seen with the infraclinoid aneurysms; but the demonstration of some objective sensory change, such as hypoalgesia in the ophthalmic division of the trigeminal nerve, favours the latter diagnosis.

Summary.

To sum up, in addition to spontaneous subarachnoid hæmorrhage, there are several clinical syndromes which should lead one to suspect an intracranial aneurysm, some of the better known being the following: (i) oculomotor nerve palsy associated with severe supraorbital pain; (ii) bitemporal hemianopia with intense headaches and a normal sella on X-ray examination; and (iii) unilateral blindness in the absence of any local disease of the eye.

The Management of Intracranial Aneurysms.

When an aneurysm is suspected, or when spontaneous subarachnoid hæmorrhage has occurred making the presence of an aneurysm tolerably certain, there is a choice in the line of treatment to be taken. Conservative measures consist of standing by and doing nothing more than performing lumbar punctures (which might merely serve to make room for further bleeding), and trusting that, if multiple hæmorrhages do not occur, the rent in the artery will seal with clot sufficiently firmly to prevent recurrence of hæmorrhage. In a proportion of cases this is quite successful, and there are many people who have had but one hæmorrhage and have continued living normally for years without further trouble. Unfortunately, there is no possible method of determining which patients will avoid further hæmorrhage and which ones will be the subjects of recurrence.

The second method available is to regard the condition as potentially surgical, which is the line recommended by the present writer. This does not mean that every patient should be subjected to operation; it implies that each patient should be submitted to angiographic examination, and, according to the site of the sac, operation may or may not be advised.

Whichever course is followed, there is a risk. If the approach is conservative, one is relying on the formation of a clot in a condition which is notorious for its tendency to recur, and in which it is well known that each recurrence has an increasing mortality rate. Ligation in the neck is by no means devoid of danger, for not every patient will tolerate occlusion of one of the carotids, be it internal or common. Hemiplegia, with or without dysphasia, may occur if the anastomosis from the opposite artery is insufficient to cope with the demands; but patients who have such accidents tend to recover to a large degree, and often completely. Lastly, craniotomy with a direct approach carries a risk with it owing to the possibility of rupture of the sac during handling, though this as a rule can be controlled, and loss of a patient on the operating table is rare. The fact remains that anyone who has an intracranial aneurysm is in dire straits, and this is even more true once the aneurysm has already ruptured. For this reason, the writer favours active measures of treatment to try to obviate further hæmorrhage. It is not illogical to ligate an artery that is bleeding, and were it not for the special position of these aneurysms, no one would argue the correctness of surgical treatment. However, interference with the blood supply of the brain carries in its train a series of hazards which must first be weighed before any drastic action can be taken.

Certain questions naturally arise. Firstly, is a surgical attack feasible in the earliest stages of subarachnoid hæmorrhage? These patients, when treated as "surgical emergencies" within a few hours of the onset of bleeding, do not seem able to withstand the strain of immediate angiographic examination and operation, and the results of this practice are bad. This is especially so when the hæmorrhage is massive and the patient gravely ill. It has been the writer's experience that, if the patient cannot survive the initial shock, surgical interference has not helped. This leads to the second question—at what stage should angiography and operation be contemplated? No unqualified answer can be given, for it depends upon the patient's resistance to the accident. The gravely ill, deeply comatose patient seems to be a well-nigh hopeless proposition, though one is often tempted, perhaps unwisely, into active measures in a forlorn desire to do something useful —and success is a rare reward under these circumstances. On the other hand, many patients survive the initial accident and begin to show clinical improvement within twenty-four hours or so. An angiographic examination should then be made as soon as the patient's general condition permits, because it is essential to forestall the recurrence of bleeding, which is particularly likely to take place within the first fortnight. The ideal is, therefore, to get on with angiographic examination within a day or two of the first hæmorrhage. It is important not to be hustled into operation too early; yet delay must be minimal, because all too often a patient who is being carefully guarded in hospital under expert observation dies under one's eyes from a massive recurrent hæmorrhage.

When it is possible to diagnose an aneurysm before rupture has occurred, the case should be regarded as urgent, for it is impossible to forecast just when the bursting point may be reached. Angiography is always an essential preliminary to surgery, even though the location of the aneurysm appears tolerably obvious by virtue of existing cranial nerve palsies; the latter are not absolute guides for, on rare occasions, though the nerve palsies may be on one side, the sac is found to fill from the artery of the opposite side.

It would be unwise to be dogmatic about the surgical treatment of individual aneurysms; throughout the world in recent years surgeons have been assessing their results with the ultimate object of gaining uniformity of treatment, but this has not yet been fully achieved. Broadly speaking, surgical treatment consists of ligation of the carotid artery in the neck, or a direct approach by craniotomy upon the aneurysm itself or its artery of supply within the cranial cavity. The sac, once it has been exposed, may be dealt with in a variety of ways—by clipping its neck, by clipping the arteries of entry and exit, or by packing muscle around as much of the sac as is accessible to reinforce its wall. The value of the last procedure is open to question unless the entire sac can be surrounded. When an indirect attack on the aneurysm is made by arterial ligature in the neck, either the common or internal carotid may be ligated and divided, ligated in continuity, or partially occluded. Both vessels may require ligation in certain cases—for example, if occlusion of the common carotid does not bring about rapid improvement,

it may be followed by internal carotid ligation after an arbitrary interval.

The essential preludes to surgery are angiography and a knowledge of the efficiency of the anastomotic circulation when any ligation of a main vessel is contemplated. The former gives the location and size of the sac, but not the total mass of the aneurysm, which depends upon the amount of solid blood clot surrounding the patent sac. It also gives information as to the multiplicity of aneurysms and to the collateral circulation (*vide infra*).

The efficiency of the arterial anastomosis can be gauged in different ways, but no method is quite foolproof. The simplest test is the compression test of Matas, which consists of digital compression of the carotid in the neck for a period of ten minutes whilst watch is kept for the development of weakness in the contralateral limbs with or without dysphasia. If no changes are observed, it is judged that the opposite carotid and the basilar arteries are able to provide an adequate supply of blood to the brain. In the performance of this test, care must be taken not to compress the carotid sinus, which may cause syncope and so confuse the issue at stake.

Angiography may be used to demonstrate the adequacy of the collateral circulation very simply. The carotid which it is planned to ligate is temporarily occluded by any convenient means, and "Uriodone" is injected into the opposite carotid. X-ray films are taken in the antero-posterior plane, and if the anastomosis is satisfactory, filling of both anterior and both middle cerebral arteries will be apparent.

Electroencephalography is also of assistance, and any disturbance of rhythm appearing on temporary occlusion of the carotid will raise suspicions as to whether its permanent ligation will be tolerated.

Finally, when it has been definitely decided to ligate the artery, the operation should be performed under local anæsthesia, and before permanent occlusion is carried out the artery must be temporarily blocked and watch kept for the development of contralateral signs. This trial ligation should be persisted with for twenty to thirty minutes, but in special circumstances some hours can be permitted to elapse before the final step is taken.

When all information has been acquired and assessed, and if the patient's general condition permits, a decision must be made about the line of attack to be adopted. Detailed discussion is beyond the scope of this paper, so that only an outline in general terms is deemed pertinent. Aneurysms fall naturally into two broad groups—those on the basilar system of arteries and those on the carotids. It is very rarely that any surgical approach can be made to the former; they are not sought by routine angiography, and there are but few reports of successful attacks made upon aneurysms in the posterior fossa. Suffice it to say here that only an occasional pedunculated aneurysm on a vertebral artery or on lesser branches of the basilar artery has proved amenable to surgical treatment.

The carotid aneurysms again fall easily into two groups —those lying on the undivided internal carotid and those arising distal to its bifurcation. The former may lie wholly within the cavernous sinus, where they do not cause subarachnoid hæmorrhage and are not relevant to this discussion; these are termed infraclinoid aneurysms. The supraclinoid aneurysms are a potent source of hæmorrhage, and included here are those on the posterior communicating artery which behave in the same fashion. These have long been treated by arterial ligation in the neck, a form of treatment which has been most satisfactory and has stood the test of time. Even so, a direct attack on some of these is made on occasions, Dandy having been the pioneer of the "trap" operation, which involved occlusion of the internal carotid in the neck as well as intracranial occlusion. Nevertheless, common carotid ligation in the neck with or without subsequent internal carotid ligation gives excellent results (Jefferson).

In the treatment of patients in whom the aneurysm lies beyond the bifurcation of the internal carotid—that is, on the anterior cerebral, anterior communicating or middle cerebral arteries—the best course to adopt is not so clear. The choice must lie between a direct attack on the aneurysm, ligation of the artery in the neck and a combination of both methods. It has long been thought that ligation in the neck would have no beneficial effect upon distally placed aneurysms, because, provided the collateral circulation coming from the opposite side via the circle of Willis was effective, the blood flow through the anterior and middle cerebral arteries on the ligated side should continue undiminished. Consequently, it was argued, there could be no lowering of blood pressure within the aneurysm and so no encouragement for clotting to occur. However, it has recently been shown by means of clinical experiments (Sweet *et alii*) that ligation of either common or internal carotid in the neck causes a considerable fall of blood pressure in many cases, so that there does, in fact, exist a good physiological argument for this form of treatment. The writer has lately reported a case with clinical and angiographic cure of an aneurysm on the first part of the anterior cerebral artery by ligation of the common carotid alone in the neck.

Nevertheless, the trend now is more toward a direct approach to these aneurysms. The greatest hazard is rupture during operation with uncontrollable hæmorrhage; but the latter, alarming though it may be, can usually be brought under control. The use of drugs and mechanical methods of creating hypotension during operation has proved a great safety measure in certain hands. Some writers have reported impressive results by the use of a direct attack both in the distal aneurysms and in those arising proximal to the division of the artery (Poppen, 1951; Falconer, 1951; Norlen, 1952).

Statistical Survey.

Intracranial aneurysms occur frequently and have been reported extensively in the literature in association with rupture. McDonald and Korb (1939) collected details of 1125 intracranial aneurysms, of which no less than 862 had ruptured to cause subarachnoid hæmorrhage. Fearnsides (1916) reported 44 aneurysms found at necropsy, of which 35 had bled; Symonds (1924) reviewed 124 instances of spontaneous subarachnoid hæmorrhage, to find ruptured aneurysm as the underlying cause in 10 cases, all ending fatally; and in Green's (1927) series of 19 aneurysms, 17 were ruptured. In 10,000 cases of violent death analysed by Martland (1939), subarachnoid hæmorrhage occurred 54 times, aneurysms being responsible in 39 instances. Sands (1941) discussed 120 cases of subarachnoid hæmorrhage of all origins, and in 25 aneurysm was the source, with 12 fatalities.

Magee (1943) produced some illuminating statistics from his study of 150 cases of subarachnoid hæmorrhage occurring largely amongst the armed forces. Fifty-two subjects (34%) died in the first attack, leaving 98 survivors, of whom no less than 50 had recurrences of bleeding with 32 deaths—that is, 64%. Thus, 84 patients died out of a total of 150, and Magee also adds that another 21 were crippled by sequelæ such as paralysis, headache or vertigo. Autopsy was carried out in 58 cases, leading to the discovery of 43 aneurysms. Hamby (1948) studied 130 cases of subarachnoid bleeding, aneurysms being proven present in 44. In this series, 98 patients were admitted to hospital in their initial attack, the death rate being 45%, whereas, of 32 admitted to hospital with recurrent hæmorrhage, 72% died.

Figures which take into account the surgical treatment of aneurysms are becoming increasingly available. Schorstein (1940) collected accounts of 60 ligations of the carotid artery, 56 having been performed for aneurysm; 22 aneurysms were in the cavernous sinus and ligation caused no deaths, though three patients developed neurological sequelæ. There were 34 supraclinoid aneurysms; 22 had not leaked, and death followed ligation in three instances; in the remaining 12 cases there had been subarachnoid bleeding, and five deaths followed ligation. These deaths occurred amongst nine patients who had had recent hæmorrhage, and ligation was undertaken as an emergency measure.

Jefferson (1947) reported 55 cases of aneurysms associated with isolated oculomotor nerve palsy, of which 40 had bled, seven cases ending fatally in hospital. He ligated the carotid in the neck 23 times, and there were three rapid deaths when operation was performed on semicomatose patients. Of the remaining patients, one died seven years later from an unknown cause and 19 remain well. Again, Jefferson (1952) referred to 250 cases of aneurysms, of which about half the number had bled. Hunterian ligature was practised 142 times; 12 patients died within six weeks, four having been *in extremis* at the time of operation. There were eight deaths at a later period, making 20 in all; in eight of these cases death was caused by bleeding from the same aneurysm. Direct attacks by craniotomy numbered 35, amongst which there were five prompt deaths. Johnson (1952), referring to the same series, pointed out that in 29 cases of severe hæmorrhage the carotid was tied within a few hours of the onset, with the survival of 17 patients, of whom but two had had hæmorrhage since.

Jaeger (1950), in a series of 31 cases, attacked 20 aneurysms intracranially with five deaths. The remaining patients are alive and well, the period of review being up to six years. Six enfeebled patients were treated by ligation in the neck, and five were not submitted to surgical treatment. Wechsler, Gross and Cohen (1951) found 17 aneurysms, 12 ruptured; all patients were treated by common carotid ligation with subsequent craniotomy in four cases. Two critically ill patients died, and a third died after late secondary craniotomy. Fourteen survived, of whom seven have been traced up to two years later and are well. Falconer (1951) published an account of 50 cases of leaking aneurysm. Surgical treatment was undertaken in all and resulted in 33 good recoveries, eight recoveries with disability, and nine deaths. Carotid ligation in the neck alone was the method employed in 27 cases, craniotomy alone in 16 cases and a combination of methods in seven. The survivors have been followed over periods ranging from six months to four years, and he reports only one recurrence. Poppen (1951) carried out carotid ligation in the neck 101 times, and death resulted three times. Another eight patients died long after operation, but in only two cases was death caused by further rupture.

Norlen (1952) gave details of 120 aneurysms found in 114 patients. One hundred and two were situated distal to the point of emergence of the internal carotid from the cavernous sinus, and 85 had bled. Thirty-seven patients were subjected to ligation of the artery in the neck, with only one death. Thirty-one of them have been traced, of whom 17 are at full working capacity and two more working hard, though epileptic. Six others have disabilities caused by convulsions, hemiparesis or mental changes; two died of other disorders and six from recurrent bleeding. Fifty-nine patients were subjected to craniotomy and a direct attack on the aneurysm, with only six deaths. Four developed hemiparesis, of whom one made a complete recovery and two partial recoveries, whilst the hemiparesis of the fourth became permanent. Of 22 patients not operated on, nine were alive up to nine years later, four died in hospital, five died within three years from renewed bleeding, two died from unrelated causes and two are untraced. Swain (1948) reported a small but instructive series of four cases of aneurysm on the middle cerebral artery supplying the dominant hemisphere. Direct attacks were made on three of these aneurysms, with most satisfactory results.

The Present Series.

The writer now submits 28 cases of proven intracranial aneurysm for analysis.

Clinical Features.

There were 15 male patients and 13 female; five were in the third decade, seven in the fourth, 10 in the fifth and six in the sixth. In seven cases there was complete paralysis of the oculomotor nerve; visual field defects were demonstrable in four, two patients having an homonymous defect and two bitemporal defects with macular involvement. Four patients were hemiplegic.

Spontaneous subarchnoid hæmorrhage occurred in 25 cases, but is not known with any certainty to have taken place in the other three. In 11 cases hæmorrhages were multiple, and in three subhyaloid bleeding could be seen in the fundi.

Pathology.

All aneurysms were saccular in type, and one only was found on the basilar artery. Twenty-seven were on the carotid system distal to the cavernous sinus, of which 14 were on the supraclinoid portion of the undivided vessel and one at the bifurcation; three were on the middle cerebral artery and nine on the anterior cerebral artery (three on the first part of this artery, four on the second part and two on the third part).

Treatment.

Four patients were not treated surgically. These included a patient with a large sac on the main trunk of the left middle cerebral artery, the risk of surgical treatment of whom was considered to be too great to be justified; another had a middle cerebral aneurysm and died before angiography was attempted; and the patient with a basilar aneurysm never presented a surgical problem. All these patients died. In the fourth, a huge mid-line sac was found at craniotomy, but as nothing effective could be done this patient is included amongst the untreated. His vision had failed and he was incapacitated by headache when last examined shortly after operation.

Twenty-four cases were treated surgically—23 by arterial ligation in the neck and only one directly. This last patient was very ill, with an aneurysm on the third part of the anterior cerebral artery which was completely excluded from the circulation at operation. Nevertheless, she died three weeks later and was found to have a congenitally abnormal circle of Willis grossly affected with atheroma, and her death was due to inadequacy of the anastomotic circulation.

Twenty-three patients were treated by carotid ligations; the common carotid was tied 15 times in continuity (11 total occlusions with silk ligatures, one total occlusion by Poppen's method and three partial interruptions with "Cellophane"), and on seven occasions ligation and division was practised. In three cases the internal carotid was ligated, twice after common carotid ligation and once as the sole operation, Poppen's method being used.

Results.

In the treatment of three desperately ill patients surgery was offered as a forlorn hope, but had no beneficial effect even of a temporary nature.

Six patients were classed as very ill indeed and were threatened with death. Four of these recovered—one completely, two with persistence of neurological sequelæ which existed before operation, and one with a residual spastic weakness of his leg, the sole residue of a total hemiplegia starting thirty-six hours after operation. The other two died—one with an aneurysm at the bifurcation of the internal carotid from renewed bleeding two weeks after common carotid ligation, the other from acute hydrocephalus caused by obstruction of the foramina of Monro by a massive mid-line aneurysm. This last man had obtained considerable relief of intractable headache and slight improvement of vision after internal carotid ligation six months previously.

Fourteen other patients affected in varying degrees of severity are all alive and, for the most part, well. Two with aneurysms of the anterior communicating artery have patent sacs and should be treated by a direct attack, though they are at present so well that the temptation is to await further hæmorrhage before advising craniotomy. Seven women are all working hard in various capacities and have minor complaints only, which cannot all be related definitely to their original condition. One other became hemiplegic sixty hours after ligation and is making a steady recovery. There are three men working, earning their living and having no complaints at all. A fourth says he is mostly unemployed, but he is an unreliable type

of individual whose appearance belies his contention, and clinically his condition is quite sound.

The Incidence of Neurological Defects.—Focal neurological signs may appear as the result of pressure of the aneurysmal sac on neighbouring structures or of injury of the brain by hæmorrhage or thrombosis, or they may follow operation. Twelve patients had focal signs before operation and have benefited as the result of operation. All seven with oculomotor palsies have made the partial recovery characteristic of these lesions—ptosis has disappeared, medial movement of the eye has recovered fully, upward and downward movement is greatly restricted and the pupil remains in the mid-position, insensitive to light. Three were hemiplegic and one has failed to return to a useful existence, though he has improved, in so far as he can now get about of his own accord, whereas he was originally densely paralysed. The other two both had an homonymous hemianopia which remains unchanged, though their paralysis has recovered and both are working usefully. One of these patients was also hemianæsthetic and had had three hæmorrhages prior to operation, so that his degree of recovery is rather remarkable. Finally in this group was a woman with severe mental disturbance, who has made a complete recovery to lead a normal domestic life once again. Four patients have neurological signs, the direct result of operation. One is a woman who had an embolus in the central retinal artery six weeks after carotid ligation. She is irreparably blind in this eye, but otherwise is perfectly well and active. This is a very rare complication of carotid ligation. Next is a young man who had a common carotid ligation after his third hæmorrhage from a middle cerebral aneurysm; dense hemiplegia developed thirty-six hours after carotid ligation; but his recovery has been of such degree that he is now earning his own living and working regularly, though his foot stays paralysed, and he requires a walking iron. A third patient is one recently treated, who developed hemiplegia sixty hours after carotid ligation. Heparin was promptly given and she recovered completely within forty-eight hours; but, as her wound then commenced bleeding, the administration of heparin was suspended, with the reappearance of hemiplegia. She is now making a slower but steady recovery. Another man developed transient hemiplegia and some sensory loss twenty-four hours after operation, but he recovered rapidly with heparin therapy, retaining only some unimportant sensory loss in a small area of the hand after two days.

Recurrence of Hæmorrhage.—Finally, one patient only died from recurrence of bleeding after carotid ligation. This was a woman whose aneurysm was situated at the bifurcation of the internal carotid; she had made excellent progress after common carotid ligation, but died suddenly on the day of her discharge from hospital. It seems probable that a direct attack should have been made on this sac, even though its obliteration by this means would have been fraught with difficulties.

Summary.

Summing up finally, there were 24 patients treated surgically, and a follow-up examination shows that 18 are alive and have had no more bleeding over periods ranging from three months to more than four years. All but three are leading useful lives, and of the latter, one is recovering from post-operative hemiplegia and should soon be capable of returning to her domestic duties, the *bona fides* of one is questioned, and there is only one who would appear to be permanently incapacitated. In criticizing the occurrence of six deaths, it must be remembered that three patients were in a desperate condition and could justifiably have been refused operation; one had a massive aneurysm beyond the scope of curative surgery, though he obtained symptomatic relief until his death from hydrocephalus; one had an abnormal circle of Willis with unsuspected atheroma; and only one died as the result of recurrent hæmorrhage.

Conclusions.

A large proportion of subarachnoid hæmorrhages are directly attributable to the rupture of an aneurysm upon one or other of the cerebral arteries. If treatment is withheld, there is a high mortality rate which, from a perusal of many statistics, would appear to be 35% to 50% in the initial attack and about 10% to 20% higher in subsequent attacks. Surgical treatment, despite certain risks, gives appreciably better results in series hitherto reported, though no great benefit is offered to those gravely ill patients who have had severe hæmorrhages with extensive damage to the brain.

The routine of surgical treatment is first to prove the existence of an aneurysm on the carotid arterial tree by bilateral angiography, which should be carried out as soon as the patient's condition permits, and, secondly, to decide what form of surgical attack should be employed. The latter depends upon the position of the aneurysm, the efficiency of the anastomotic circulation and the general condition of the patient, and arterial ligation in the neck may be chosen or, alternatively, a direct intracranial attack upon the aneurysm itself.

It is the writer's opinion that an angiographic examination should be made on all patients who survive the initial hæmorrhage, and should an aneurysm be revealed on the carotid system, surgical treatment should be seriously considered and discussed with those concerned.

Summary.

A brief historical introduction precedes an account of the clinical features of intracranial aneurysms and spontaneous subarachnoid hæmorrhage.

The management of these conditions is described from the surgical aspect, and statistics from numerous papers are quoted with especial reference to surgical treatment.

Details of 28 new cases are discussed, in 24 of which surgical treatment was given with recovery in 18.

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THE POLAROGRAPHIC CANCER TEST.

By F. OFNER,
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IN 1934 Purr and Russell found that blood serum of cancerous patients did not stimulate the proteolytic activity of papain and kathepsin in the same manner as does serum of normal subjects. This enzyme-stimulating property of normal serum was ascribed to sulphhydryl groups (SH groups) present in them. It is Brdicka's merit to have found a method for the determination of SH groups by polarography.

Polarography is an electrochemical method for micro-analysis. It consists in applying a steadily increasing direct potential to a dropping mercury electrode and recording the current flowing through the electrolysis cell at various voltages. Wave-shaped current-voltage curves (polarograms) are thus obtained, whose height depends on the concentration of the substance undergoing the electrochemical change. The concentration range most commonly used for polarographic work is from 10^{-3} to 10^{-5} molar. The polarographic estimation of SH groups is based on the fact that a new wave appears in the polarogram when compounds containing SH groups are added to a cobalt salt solution (Figures IA and IB).

The Polarographic Cancer Test.

For the polarographic cancer test, serum is partially hydrolysed with potassium hydroxide in the cold. The native proteins are then precipitated with sulphosalicylic acid, and the filtrate containing the proteoses is subjected to the polarographic SH group test. Generally the SH group concentration is found higher than normal in the filtrate from cancerous serum.

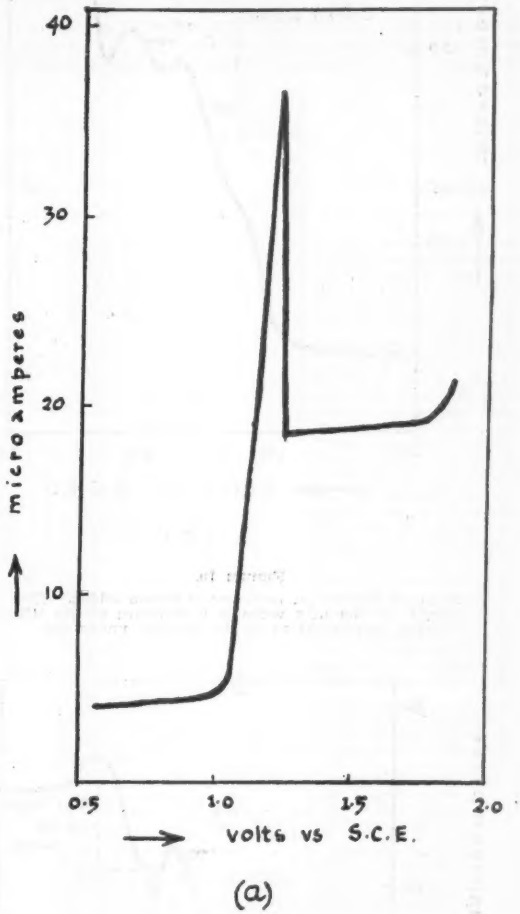

FIGURE IA.
Polarogram of the cobalt buffer.

The use of the polarographic method for the diagnosis of cancer has been rather controversial (Brdicka, 1937; Tropp, 1938; Bergh *et alii*, 1938; Albers, 1940). Different variations of the polarographic technique have been evolved. The one adopted by us follows that recommended by A. Robinson (1948).

Amounts of 0·4 millilitre of blood serum are treated with one millilitre of N/10 potassium hydroxide solution, kept at room temperature for forty-five minutes, and the proteins are precipitated by the addition of one millilitre of a 20% sulphosalicylic acid solution. The mixture is allowed to stand for exactly ten minutes and filtered through Number 50 Whatman paper. Of filtrate 0·5 millilitre is added to five millilitres of a buffer consisting of M ammonia, 0·1 M ammonium chloride solution and 0·001 M cobaltihexamine chloride solution. To remove oxygen, hydrogen gas is passed through the solution for ten minutes.

As is shown in Figures IA and IB, the polarograms obtained consist of two waves: the cobalt wave produced by the reduction of the cobalt ions of the solution, followed by

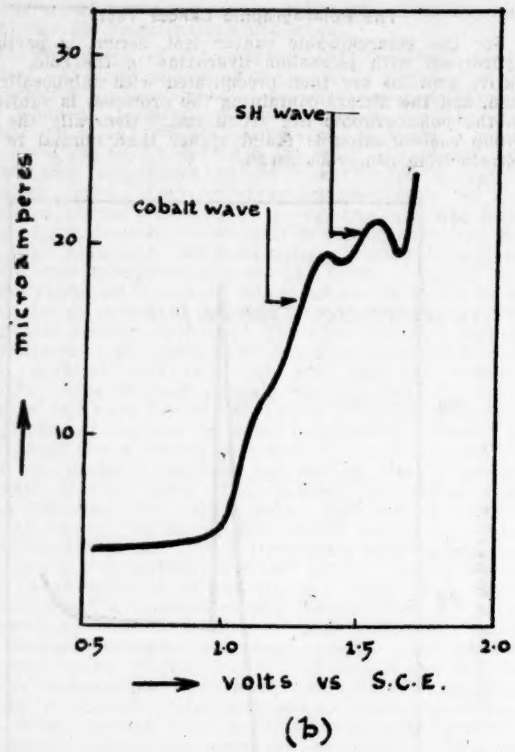

FIGURE Ib.

Same as Figure Ia, but normal serum added. The height of this new wave is a measure of the SH group concentration in the solution under test.

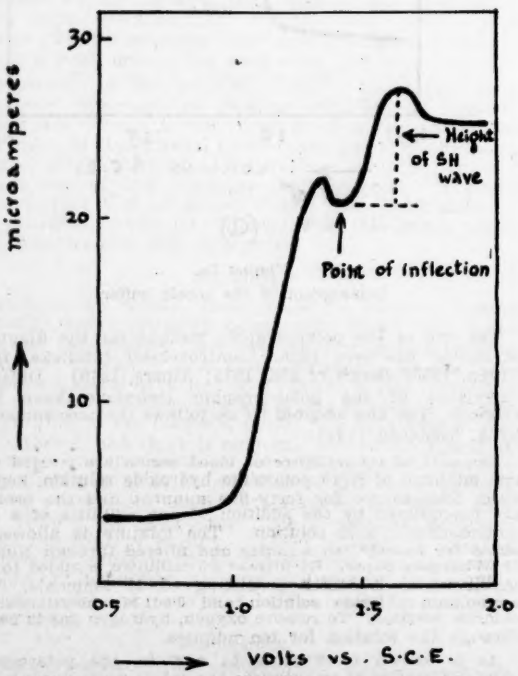

FIGURE II.

another, the relevant wave, produced by the presence of SH groups. Robinson measured the peak height of the wave, taking the point of inflection between the end of the cobalt wave and the beginning of the filtrate wave as the base line (Figure II).

In our experience, however, this way of measuring is not adequate. We found that high polarographic waves—that is, waves characteristic for cancer serum—can be grouped in two classes; one class shows an increased SH wave preceded by a cobalt wave of the same height as that found in normal serum, whereas in another group of serum from cancer patients, both the SH wave and the cobalt wave are increased. In the latter case the absolute height

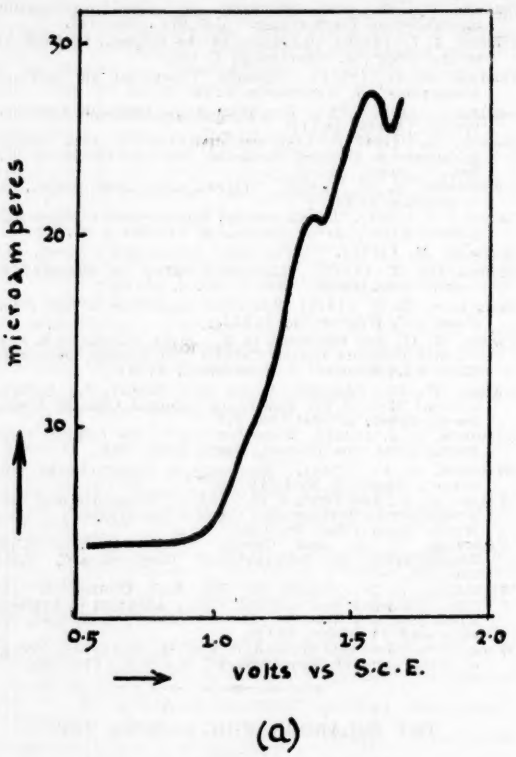

FIGURE IIIA.

Relative positive polarogram.

measured from the beginning of the step is considerably raised. We propose to call the first type of wave a "relative positive polarogram", the second type an "absolute positive polarogram" (Figures IIIA and IIIB).

We cannot ascertain the importance of this finding, but we think it is worth while mentioning it for the sake of further investigations, because we found that in cases of prostate cancer, cancer of the ovary and some cancers of the breast, as well as in most cases of cancer of the pancreas and the lung, absolute positive polarograms were obtained, whereas in cases of malignant tumours of other organs together with a number of non-specific inflammatory processes, a relative positive polarogram was generally obtained. We should also like to emphasize that there seems no strict correlation between acid and alkaline phosphatase values and abnormal polarograms, a fact already noted by Robinson.

Discussion.

The difficulty in using polarography as a specific cancer test arises from the fact that similar polarograms are obtained in cancer as well as in all acute inflammatory

conditions. The best way to exclude the possibility of an acute, non-malignant condition is to repeat the test in a fortnight's time and to see whether the polarogram shows a tendency to return to normal. Fortunately, as a rule, chronic diseases do not affect the polarographic wave height and thus do not interfere in the diagnosis of malignant tumours.

Two cases of hepatic involvement are worthy of mention:

CASE I.—The patient, a male, aged seventy-five years, was suspected of having either gall-stones or some malignant disease causing obstruction. The serum bilirubin content was 7·0 milligrammes per 100 mils, the thymol turbidity was 2·0 units, and the serum alkaline phosphatase content was 15 units. Polarography resulted in the production of a relative positive wave. This inconclusive result was clarified after twelve days; the polarogram returned to normal, and the condition was finally diagnosed as biliary obstruction caused by gall-stones.

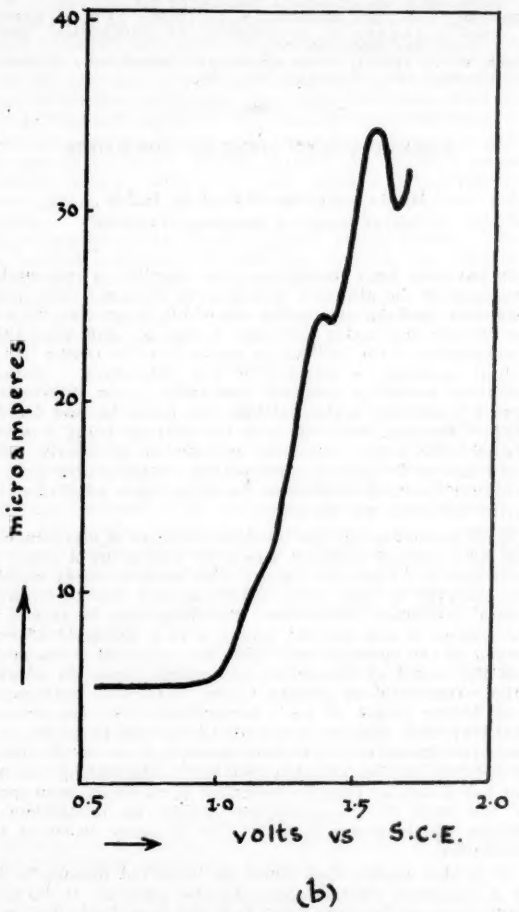

FIGURE IIIB.
Absolute positive polarogram.

CASE II.—The patient, a male, aged sixty-three years, had abdominal pain and diarrhœa and had been jaundiced for five weeks. The serum bilirubin content was 17 milligrammes per 100 mils, the thymol turbidity was 1·5 units and the serum alkaline phosphatase content was 42 units. In the polarogram a relative positive wave was produced. At operation cancer of the pancreas was found. Twelve days after operation the polarogram showed no tendency to return to normal.

The polarograms obtained in acute hepatitis warrant specific mention. In two cases of infective hepatitis the thymol turbidity test gave a positive result, but the polarogram showed an increased wave in only one case, thus also revealing a discrepancy between thymol turbidity values and positive polarograms. This discrepancy was also shown in other cases of liver involvement—for example, in a non-malignant hepatic condition with a thymol turbidity value of six units. In this case the polarogram was almost normal. In a case of pancreatic tumour, however, a positive wave was found together with a thymol turbidity value of only 1·5 units.

In a certain number of jaundice cases, with high serum bilirubin values, the cobalt wave was completely suppressed. This was a rather awkward circumstance, as in these cases the point of inflection between the end of the cobalt wave and the beginning of the SH wave had disappeared, and it proved impossible to measure the height of the second wave (Figure IV).

TABLE I.
Cases Investigated.

Code Number.	Diagnosis.	Type of Polarogram.
1	Cancer of breast.	Absolute positive polarogram.
2	Cancer of breast.	Absolute positive polarogram.
3	Cancer of breast.	Absolute positive polarogram.
4	Paget's disease of the breast.	Absolute positive polarogram.
5	Cancer of ovary.	Absolute positive polarogram.
6	Cancer of ovary.	Absolute positive polarogram.
7	Cancer of ovary.	Absolute positive polarogram.
8	Cancer of prostate.	Absolute positive polarogram.
9	Cancer of prostate.	Absolute positive polarogram.
10	Cancer of prostate.	Absolute positive polarogram.
11	Cancer of prostate.	Absolute positive polarogram.
12	Cancer of prostate.	Absolute positive polarogram.
13	Cancer of prostate.	Absolute positive polarogram.
14	Cancer of prostate.	Absolute positive polarogram.
15	Cancer of lung.	Absolute positive polarogram.
16	Cancer of lung.	Absolute positive polarogram.
17	Cancer of pancreas.	Absolute positive polarogram.
18	Cancer of pancreas.	Absolute positive polarogram.
19	Cancer of pancreas.	Absolute positive polarogram.
20	Cancer of pancreas.	Absolute positive polarogram.
21	Cancer of pancreas.	Absolute positive polarogram.
22	Cancer of pancreas.	Absolute positive polarogram.
23	Cancer of pancreas.	Absolute positive polarogram.
24	Cancer of vagina.	Relative positive polarogram.
25	Cancer of intestine.	Relative positive polarogram.
26	Cancer of intestine.	Relative positive polarogram.
27	Cancer of intestine.	Relative positive polarogram.
28	Cancer of intestine.	Relative positive polarogram.
29	Cancer of pancreas.	Relative positive polarogram.
30	Cancer of pancreas.	Relative positive polarogram.
31	Cancer of prostate treated with stilbœstrol.	Relative positive polarogram.
32	Infective hepatitis.	Relative positive polarogram.
33	Infective hepatitis.	Relative positive polarogram.
34	Subacute nephritis.	Relative positive polarogram.
35	Cancer of bladder.	Relative positive polarogram.
36	Cancer of stomach.	Negative polarogram.
37	Cancer of stomach.	Negative polarogram.
38	Fibroadenoma of breast.	Negative polarogram.
39	Obstructive jaundice — gall-stones.	Negative polarogram.
40	Cholecystitis—gall-stones.	Negative polarogram.
41	Infective hepatitis.	Negative polarogram.
42	Muscular rheumatism—fibrositis.	Negative polarogram.
43	*Diabetes mellitus.*	Negative polarogram.
44	Chronic arthritis.	Negative polarogram.
45	Hypertrophy of prostate.	Inconclusive polarogram.

From available data and references, as well as from our own limited material, we may conclude that there are two groups of malignant tumours—one producing changes in the number of SH groups in the serum proteins and thus positive waves in polarograms, and the second group with negative waves—that is, with unaffected SH groups. The latter seems to be the case especially in tumours of the gastro-intestinal tract, the buccal cavity and the skin. Butler (1951) found positive results in only 10 of 19 cases of malignant tumours of the buccal cavity and the skin, whereas of 55 cases of cancers of other organs (excluding cancers of the pharynx, larynx and buccal cavity), in 51 a positive result was obtained. In our own experience, gastric cancer gave negative polarographic findings.

In conclusion, we can say that the polarographic method seems to have a special value in the differential diagnosis of prostatic cancer as well as of cancer of the pancreas. Polarography seems to be most useful in the prognostic evaluation of cancerous conditions. Patients with malignant tumours who give positive polarographic findings and undergo operation should show a return to normal within approximately a fortnight after operation. If this is not the case, one can assume that metastases are present, or a hidden primary tumour. However, if the polarographic findings return to normal after the operation, regular polarographic investigations at intervals of approximately six months, together with other routine tests, can supply a very useful and early means for the detection of any prognostically unfavourable change.

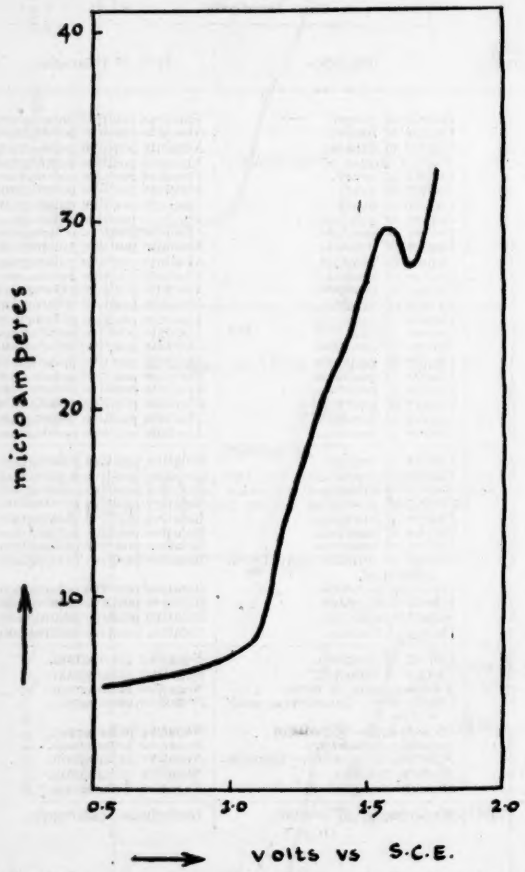

FIGURE IV.
Type of cobalt wave occasionally found in serum with an excess of bilirubin.

Summary.

1. The application of polarography as a diagnostic aid for the detection of malignant tumours is discussed.

2. It is shown that two different types of polarograms are frequently obtained, according to the location of the tumour.

3. The value of polarography as a prognostic aid and for the early detection of metastases is stressed.

Acknowledgements.

I wish to thank Dr. B. Breyer, University of Sydney, who suggested this investigation, and under whose supervision the work was carried out. My thanks are also due to Dr. Marjory Thomas, of the Pathology Department, Manly Hospital, for her support and advice, to the technical staff for their cooperation, and to Dr. E. A. Hedberg, Sydney Hospital, for checking the manuscript. The investigation was made possible by financial assistance from the Manly Hospital Board. For the drawings I am indebted to Mr. S. Hacobian, M.Sc., University of Sydney.

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HEREDITY AND MENTAL DISORDER.

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IT has long been recognized that heredity is responsible for many of the ailments which occur in man. The great dramatist Shakespeare makes one of his characters refer to the various infirmities that man is heir to; and more than a suggestion of the hereditary factor is to be traced in the mental make-up of several of his characters. Hamlet doubtless owed his schizoid symptoms to an inheritance from his mother; while Caliban, the imbecile, had for his mother Sycorax, referred to in the play as being a witch. Possibly the great dramatist anticipated by nearly three centuries Mott's law of anticipation, which states that in hereditary mental conditions the offspring is affected at an earlier age than the parent.

Much knowledge of the hereditary nature of many mental and also general diseases has been gained by a study of uniovular and binovular twins. This mode of study enables the observer to note what conditions are due to environmental influence. More exact knowledge can be gained by the mating of one affected person with a similarly affected person of the opposite sex. This has occurred occasionally and the result of the union has always been an affected child. One child at present in the Children's Cottages at Kew is the result of such a marriage. In the case of phenylpyruvic oligophrenia, which is known to be due to a recessive characteristic in both parents, never by design or by accident has the problem been put to the test by mating; nor has a similar test, by design or by accident, been made in the case of the condition known as mongolism—a disease which occurs in about one in every 10,000 of the population.

It is also known that when an inherited disease is due to a recessive characteristic in the parents, it is more likely to be severe than when it is due to a single dominant characteristic in one of the parents. Much exact knowledge has been gained from the study of phenylpyruvic oligophrenia, because even the mildest case can be recognized by the excretion of phenylpyruvic acid in the urine.

That mongolism is due to heredity is to be presumed from a consideration of the figures available from the records of the Children's Cottages at Kew over a period of many years. Few of the affected children have been the last of a long family or the result of the union of relatively aged parents. Most have been the first, second or third child; and in most cases the parents have been young. Cases have occurred more than once in a family. The first and second children, and in other instances the first and third children, have been mongols. In one case in which binovular twins were born, one was normal and the other was a mongol. In one case uniovular twins, who were identical females, were both mongols. Mongols can be fertile. In one case, when a mongol male was married to a normal

female, the only child was a normal male. The writer has heard of a case in which a normal male was married to a mongol female and the children were normal.

Another interesting case history is attached to a mongol child admitted to the Children's Cottages. The father by his first wife had normal children. He divorced her and remarried. By his second wife he had four children, three of whom were mongols. His first wife remarried and, it is stated, has had normal children.

Schizophrenia in Identical Twins.

Two sisters, now aged twenty years, are at present patients in the Mental Hospital, Sunbury. They are identical in complexion, in colour of the eyes and hair, in look almost exactly alike. The condition can most correctly be described as juvenile schizophrenia, but has also been called schizophrenia on a mental deficiency basis. Owing to a degree of periodicity in the exacerbation of symptoms these subjects are sometimes diagnosed as suffering from manic-depressive psychoses; but these conditions constitute a well-recognized group of the schizophrenic disorders.

The attacks of schizophrenia came to be noted first about the age of puberty. During attacks the patients become restless, erratic, inattentive and noisy. Grimacing and gesticulation occur in both, and there are various mannerisms. They are frequently violent during attacks. Other characteristics noted include resistiveness. When the excited condition subsides the patients are relatively simple and apathetic. Hallucinations have not occurred. The general health of both has been good. There is a sister, who resembles the twins in general features; mentally she is normal.

Discussion.

Bruno Lustig (1950) has reported a case of identical mental depression in identical twins. These were almost identical in complexion, in colour of the eyes and hair, in blood groupings and in various other characteristics, and their speech, gestures and gait were such that people could not tell them apart. Their medical histories were alike, and they suffered from recurrent mental depression.

In the present instance the identical twins have the same or closely similar physical conditions, and their personality traits, manner of speech, kind of voice and general behaviour have always been alike. It is suggested that an hereditary characteristic is responsible, possibly caused by a single gene.

Summary.

Cases of mental disorder have been described. It is suggested that they throw some light on the relation between heredity and mental disorder. As in animal genetics, it is presumed that exact knowledge regarding the hereditary nature of some of these conditions can be obtained only from rare examples of breeding out or mating of affected persons with similarly affected persons.

Acknowledgement.

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INFECTIVE HEPATITIS: A DISCUSSION ON ITS MODE OF SPREAD IN FAMILIES.

By D. J. R. SNOW,
Perth.

"... The disease has an astonishing behaviour in families ... which has no counterpart in any other disease." (Pickles, 1939.)

FAMILIES may be regarded as epidemiological units.

The behaviour of a transmissible disease within family groups is instructive of its epidemiology. It also contributes to the overall pattern of that disease in a community. The larger the family, the more revealing will be this behaviour; and the greater the number of such groups, the more significant will be the effect upon the general pattern. It is perhaps regrettable that opportunities of observing the spread of disease in large families are no longer numerous. Families have shrunken greatly in size. Nevertheless, even today the family is a useful vantage point from which the epidemiology of a disease may be viewed.

Infective hepatitis is a disease in which more than one case in the same household is not uncommon. Among 1000 recorded cases in Western Australia, there have been 31 such examples (comprising 79 cases). Instances of five or more clinical infections in the one family are, however, by no means common. Two Perth families in which all seven members were affected, and one in which only the parents escaped, have therefore been the subject of special study, and the findings are here presented.

Each of the homes concerned was visited, and the patients and relatives were carefully interrogated. When necessary, additional information was sought from the doctor concerned. The dates of onset are shown in the time charts, and the probable sequence of infection in each family is indicated in the accompanying diagrams (Figures I to III).

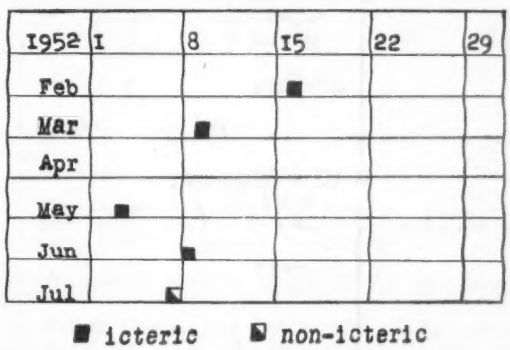

FIGURE I.

Family *A*, time chart. Mother and father, both aged forty-two years, both well; all five children affected.

The family *A*, which included five children ranging in age from two to fourteen years, harboured the infection for approximately six months. The straggling occurrence of individual infections is quite evident. The intervals between the respective onsets ranged from twenty-one to forty-two days. The parents escaped, and the youngest children were the last to be affected. Despite a low income and a small house, the standard of domestic management and personal hygiene was very high.

In the family *B*, which was equal in size and of approximately the same age structure as *A*, the infection swept through the entire household in less than three months. The intervals ranged from nineteen to forty-three days. Both parents suffered, and the youngest children were affected early. Instead of a long-drawn-out succession of cases, there was a conspicuous "secondary wave" consisting of at least three, and probably four cases, which is suggestive of simultaneous infection. The home was sub-standard and the level of personal hygiene below average. Opportunities for the exchange of infection would have been frequent and numerous. A point worthy of mention is that the wife would seem to have infected the husband.

In the family *C*, all seven members were affected in exactly two months. The intervals varied from twenty to thirty-six days. There was a pronounced "secondary wave" of four cases suggestive of simultaneous infection. Both parents were involved, and the youngest children suffered early. The home was large but ill-kept, and the standard of personal hygiene in the family was unsatisfactory. A noteworthy feature is that the husband appeared to have

infected his wife. Pickles (1939) has remarked that it is the female partner that seems to infect the male rather than *vice versa*.

Discussion.

At the outset it is well to recall two features which are common to members of the same family living under the one roof. They share a greater number of like genes than do casual persons in the community; and they are in constant and close contact with one another. There is, therefore, likely to be comparable susceptibility to specific disease, and when a communicable disease is introduced into the home by any one member it is likely to be transferred to other non-immune members of that family.

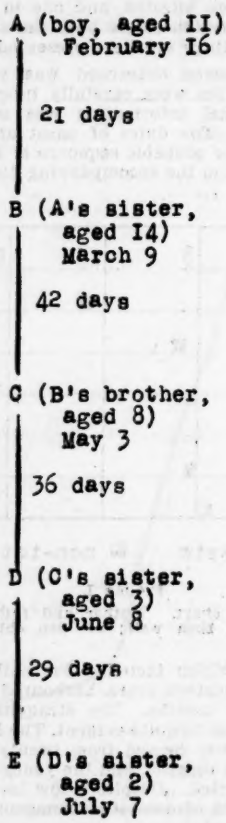

FIGURE II.

Family *A*, probable sequence of infection. Small, very well kept home, low income group; assessment of personal hygiene, "excellent".

One of the known methods of spread of infective hepatitis (and perhaps the commonest in civilian outbreaks) is by personal contact. The intimacy of familial contact should therefore be highly conducive to its spread. McFarlan (1951) reported that "the secondary attack rate at all ages in persons exposed to a case at home was twice the attack rate in the village as a whole, and the attack rate in adults exposed at home was five times that in adults in the village".

A highly infectious disease, when introduced into a family group by a child infected at school or elsewhere outside the home, readily affects the siblings. A wave of secondary cases involving all non-immunes soon follows. Such, however, is not the characteristic pattern of infective hepatitis. A straggling succession of cases spaced at long and irregular intervals is much more typical. Pickles (1939) has applied the term "serial incidence" to this phenomenon. The term "kangaroo sequence" may, however, be more appropriate, because the infection seems to skip irregularly through a family. McFarlan (1945), in his study of familial cases in English villages, found that the

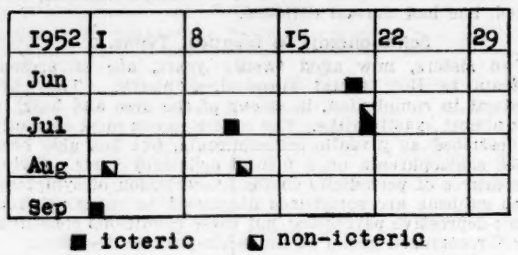

FIGURE III.

Family *B*, time chart. Mother, father and five children all affected.

cases following the first tended to occur in three waves, the first wave consisting of parallel primary cases, the second wave about a month later, and a third wave apparently due to infection by some intermediate case.

Although personal contact is widely acknowledged as a method of spread, the precise mechanism involved has not yet been determined. There are two main possibilities.

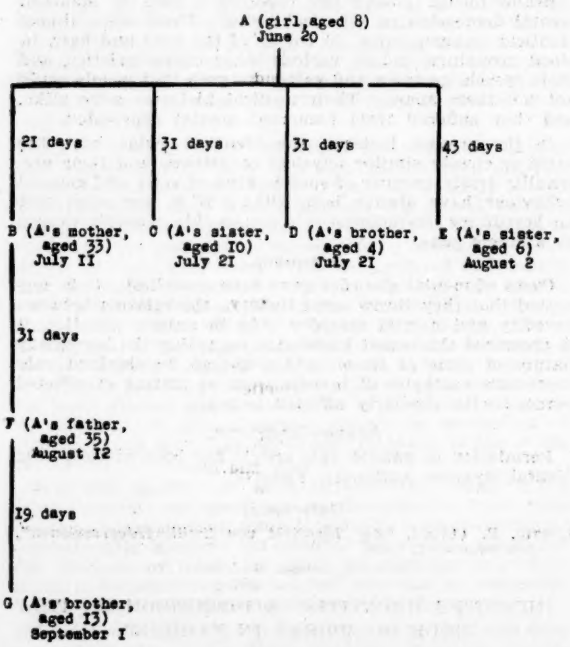

FIGURE IV.

Family *B*, probable sequence of infection. Small, very old, substandard home. Lowest income group; assessment of personal hygiene, "poor to fair".

Each has alternatively been emphasized. The first is transmission by oro-pharyngeal or naso-pharyngeal droplet, as is already known to occur with some of the common virus diseases of childhood. This hypothesis receives support from several observations. Thus, the seasonal incidence of the disease is consistent with droplet spread. Occasionally, brief contact is sufficient to allow infection to be acquired.

The disease sometimes presents like an upper respiratory tract infection, with a sore throat and a slight cough. Finally, it is the opinion of several observers, who have investigated certain epidemics, that spread in these was by droplet. Nevertheless, all this evidence is essentially circumstantial, and the virus is not readily recoverable from naso-pharyngeal washings. The evidence in favour of spread by fæcal transference is stronger. The virus can be consistently demonstrated in the fæces of affected persons. The disease can readily be reproduced by feeding infected fæcal infiltrates to volunteers. Some war-time epidemics have coincided with undue fly prevalence, and others have been associated with outbreaks of known intestinal diseases. The contamination of drinking water

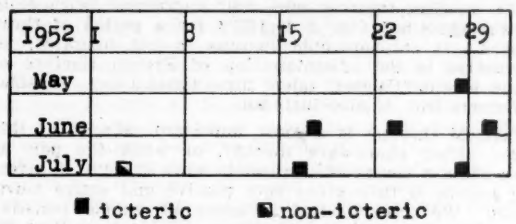

FIGURE V.

Family *C*, time chart. Mother, father and five children all affected.

with fæces has apparently been responsible for several outbreaks. The disease itself usually presents with alimentary disturbances such as nausea, vomiting and abdominal discomfort. Finally, and as a rule, close contact seems to be necessary for transmission. In fact, it may be more proper to regard this disease as contagious rather than infectious.

It is clear that the living conditions and social habits of a given family must influence the spread of communicable disease within it.

Droplet infection is associated with the acts of speaking, coughing and sneezing, and the risk increases in that order. Where there are inadequate living space, defective ventilation and poor personal hygiene, the spread of infection will be facilitated.

Infection by so-called "fæcal transference"—that is, intestinal-oral, or "hand-to-mouth" infection—is directly related to individual hygiene. When cleanliness at toilet and hand-washing are perfunctory, opportunities for the spread of infection will be increased.

Both mechanisms of disease transmission will also be influenced by the age structure within a family. Infants are ignorant of the social niceties. They may, therefore, be a source of infection. On the other hand, they are largely dependent and lead fairly sheltered lives protected from infection from elder siblings. As age advances, children become less dependent and more adventurous, but their sense of personal hygiene is slow to develop and requires constant supervision. They tend to sneeze and cough unguardedly, to play intimately with each other, and to handle each other's toys; they seldom cleanse themselves satisfactorily at toilet, and tend to regard hand-washing as a nuisance to be avoided. In short, they give and receive infections freely. These considerations probably account for the relatively high prevalence of communicable diseases of all kinds in the five to nine years age group.

Adolescents and young adults, by reason of their improved personal hygiene and increased social consciousness, are less likely to convey disease in this way, but will be exposed to infection from younger siblings.

Thus, opportunities for the spread of disease by both fæcal and droplet mechanisms will be maximal in the young school-child, but highly variable with infants and adults.

In other words, the epidemiological pattern of communicable disease in a family may be determined by the relative ages of the various members, and more particularly by the number of children in that family who are between the ages of five and ten years.

So far as the disease itself is concerned, the anatomical location of the causative agent, its duration in that location, its infectivity, the site of implantation, the incubation period of the disease and its symptomatology will all have a bearing on the ultimate character of its epidemiology.

The behaviour of a communicable disease in a family is therefore the resultant of many variables, so that a number of different patterns is possible.

On the one hand, all members may be affected; on the other, all but one may escape; while the variety of inter-

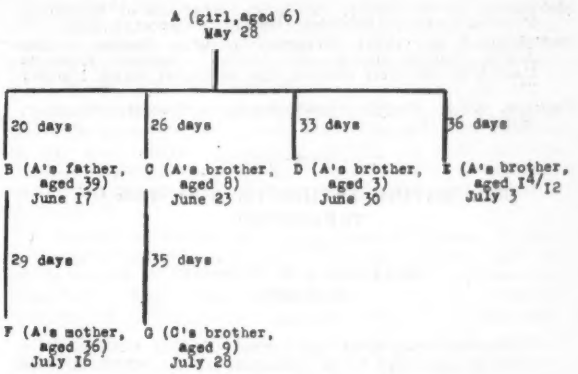

FIGURE VI.

Family *C*, probable sequence of infection. Large but ill-kept home, medium income group; assessment of personal hygiene, "poor to fair".

mediate experiences will be limited only by the size of the family.

A further complication is that the severity of virus diseases often tends to increase with age. In very young children these diseases are often so mild that they may go unrecognized. Hence, the need for simple precautions is not appreciated. On the other hand, the florid disease in older children is easily identified, and it is usual for some preventive measures to be applied.

The communicability of infective hepatitis appears to be low.

A droplet disease such as epidemic influenza is transmissible through brief exposure "by proximity" rather than by direct contact. On the other hand, a true contact disease such as leprosy requires prolonged and intimate contact for the transference of infection. Typhoid fever requires contact sufficiently close for contamination of the fingers, although the duration of this contact may be quite brief. Infective hepatitis has something in common with all these diseases.

Its period of infectivity is uncertain, but it evidently requires closer contact than is necessary for the transmission of most virus diseases known to be spread by droplet. Proximity does not appear to be enough. The nature of the contact, when contact can be established, is usually close and fairly intimate—such as that between brother and sister, husband and wife, nurse and patient, close friends, playmates and the like. In other words, the type of contact which is responsible for the transference of infective hepatitis from person to person is very much closer than is usual with the common exanthemata.

All these features are consistent with the transference of infection within families through the agency of fæcal particles, rather than by the droplet mechanism.

Summary and Conclusions.

Consideration of the behaviour of infective hepatitis in families is of assistance in appreciating its epidemiology.

The factors which influence this behaviour include standard of personal hygiene, size of family and age grouping of its members. The pattern of infective hepatitis in large families suggests that close and intimate contact is necessary for infection, and that the mechanism of transmission is probably transference of particulate fæces from the affected to the susceptible.

Acknowledgement.

This communication is published with the permission of Dr. Linley Henzell, Commissioner of Public Health in Western Australia. I am indebted to Mr. A. C. Waldon for the charts.

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RHEUMATOID ARTHRITIS: A SYSTEM OF TREATMENT.

By CLARENCE M. MARSHALL,
Melbourne.

> Perfectionists need not bother treating patients with chronic arthritis. The outcome of any effort directed towards the management of this disease is, at times, far short of the ideal. Improvements are relative. The best one can hope in chronic arthritis is some gain in useful movements, a measure of stability, or perhaps surcease from pain. But what may seem an insignificant benefit to a well person is often a major boon to one afflicted with a crippling disease. However gnarled and knotted, the patient must not be denied his chances to better his lot. (Kelikian, 1949.)

THIS statement, written by a noted American orthopædic surgeon in 1949, has more significance today, because with the cooperation of physician, orthopædic surgeon and physical therapist, a great deal can be done to help the lot of rheumatoid sufferers.

Since the introduction of cortisone and corticotropin (ACTH) for the treatment of rheumatoid arthritis, we have had placed in our hands by Kendall and by Hench and his co-workers the most useful research tools ever developed for the study of rheumatic and certain other diseases.

I have had the opportunity of studying closely the results of administration of cortisone during the past two years with a follow-up of 65 patients whose ages range from fifteen to sixty-eight years. It was realized at the outset of treatments in September, 1950, when I first commenced using cortisone, that a somewhat modified plan would have to be used from that which I observed in a large number of American clinics.

The first consideration was that the cost of this hormone, as well as its initial scarcity of supply in Australia, necessarily modified the course of treatment, which has been individualized to suit the needs of each patient. Therefore, a plan of interrupted administration has been adhered to, which is an alternative method at present adopted by Dr. Hench and his colleagues at the Mayo Clinic (Hench, 1951). It cannot be too strongly stressed that by far the largest group of rheumatoid sufferers do not require cortisone or ACTH, and in relation to those given these hormones, the most important observation made is the correct choice of patient. This will be discussed below.

Therefore, if cortisone or ACTH is not the remedy of choice for rheumatoid arthritis and its allied conditions, what is? The answer to this question forms the basis of this survey. The opportunity offered during an extensive visit to many American clinics two years ago served to permit me to devise a progressive programme for the treatment of rheumatoid arthritis. It is dependent upon the severity of the disease.

Programme I.

The treatment for early or mild rheumatoid arthritis, with little progression, consists of mainly general measures with special attention to details. A complete physical examination together with detailed blood examination is most important. A search is made at this stage to exclude infected foci, in which X-ray examination of teeth and antra is not to be overlooked. If the examination has revealed a focus, it is removed, and this is followed if possible by a course of autogenous vaccine, or Warren Crowe vaccine, together with half a gramme twice daily of "Proseptasine" ("M & B 125") for a period of three months. If sulphonamide therapy is not tolerated, an alternative is the administration of French tincture of iodine, or one "Calsiod" tablet three times a day. A tablet of ferrous iron is also included.

Physical therapy is a most important adjunct at this stage; either short-wave therapy, or when the pain is more acute a course of ultra-sonic wave therapy is given. The patient is then given both passive and active movements. We have devised a series of simple resistive exercises for active arm and leg movements, as well as for the trunk. For example, an old used bicycle tube is fixed by means of a table or the leg of a chair, and the patient is told to straighten his or her leg against the resistance of the rubber tube. If the tube is too strong to overcome resistance movements, the tube may be cut longitudinally in half. The patient is told to practise the exercises at home. A useful arm and shoulder exercise is carried out with a small weight raised by means of a rope over a simple pulley fixed to a wall. The weight may be increased as desired. Patients become very interested in these active movements, and this interest is a great help in the manipulation and passive stretching of joints which are beginning to become fixed in varying degrees of flexion.

Reduction of articular trauma is achieved by applying plaster of Paris casts or splints to the affected joints. This measure gives relief and reduces the amount of analgesics used. In inflammatory or rheumatoid arthritis, it is especially important that the joints shall be splinted in the position of optimum function should ankylosis intervene. It is often possible to obviate ankylosis by making the cast removable so as to permit periods of guarded exercises.

I have found a most useful analgesic, which consists of a 20% solution of amino-phenazone (dimethylaminophenyldimethyl pyrazolon), together with "Novocain" (2%) in normal saline solution. This preparation may be prepared by any dispensing chemist and is given in amounts of three millilitres; injected intramuscularly twice or three times weekly in the gluteal region, it considerably minimizes pain. There are no side effects. It is my practice never to give opiates. Aspirin with combinations of tincture of gelsemium and the like is most useful in the control of pain.

Proper nutrition is advised, and when there is obvious loss of weight with anorexia, two injections per day of five units of soluble insulin have quickly restored appetite. The patients must be examined frequently, at least three times a week, particularly for physical therapy.

Should this programme prove inadequate after reasonable trial, then other treatment is commenced.

Programme II.

The second programme consists of the giving of cortisone or ACTH.

The plan of administration should fit the medical requirements and the clinical characteristics, finances and psychology of each patient.

The desirable clinical results depend largely on the size of the total daily dose. Likewise some of the undesirable physiological effects are chiefly related to the daily dose. It was found that of the 65 patients treated by cortisone

only two exhibited signs of hypercortisonism, and on reduction of the dose the side effect in each instance disappeared.

In general, initial suppression of symptoms was accomplished by 75 milligrammes *plus* or *minus* about 25 milligrammes per day in divided doses, and suppression was maintained by a daily dose of about 40 milligrammes *plus* or *minus* 12·5 to 25 milligrammes of cortisone.

Proper spacing of dosage through the day is essential; cortisone was given three times a day preferably, either in tablets by mouth, or diluted with an equal part of syrup of wild cherry when at times the tablets were not available. Oral administration was 99% effective. This programme gave prolonged optimal (but not necessarily maximal) relief without pronounced exacerbations. The cortisone therapy was continued for periods of about five to six weeks, with periods of rest up to three months before cortisone administration was resumed. During the course of cortisone, injections of three millilitres of aminophenazone solution, as described above, were given twice a week. Physical therapy is begun about one week after cortisone therapy has been commenced. After about six weeks of combined treatment, administration of the hormone is discontinued and exercises are continued. Four subjects in this group had reactions and cortisone therapy was commenced again; an average maintenance dose was about 50 milligrammes a day.

It has been found that when physical therapy is first begun, passive stretching of muscles and joints, the application of casts and manipulation also are indicated. Then by the institution of walking with various aids, and by resistive exercises, in the manner described by W. B. Snow and J. A. Coss (1952), according to the ability of the patient, it becomes possible to increase range of movement and muscle power. The combination of hormone therapy and physical measures gives great relief of pain, and rehabilitates severely handicapped persons who I consider would not be helped by either measure alone.

A discussion is convenient at this point on the side effects of cortisone, and this controversial subject is dealt with most adequately by Hench in his paper delivered at the Conference on the Effects of Cortisone, held at New York on December 10, 1951 (Hench, 1951). Concerning side effects of cortisone Hench makes the following statement:

> It is not surprising that many physicians and patients fear these unknown, indefinite, hypothetic side effects more than they fear the side effects which have been studied so intensively and described in so much detail.
>
> With all due regard to their potential importance I believe that both the real and the hypothetic side effects have been considerably exaggerated in the medical and lay press, not only by conscientious physicians but also by deliberate alarmists and publicity-seeking lay writers who "had to exaggerate" in order to sell a story and rouse interest. An objective appraisal of both the undesirable and the desirable effects of these new hormones is certainly needed; each should be studied carefully in the light of the other.
>
> The development of a healthy sense of respect for these new, powerful hormones is very important, especially for physicians who simply do not have sufficient time to manage their patients carefully.

Hench goes on to describe the tragic suicide on December 31, 1948, of a successful American writer suffering from rheumatoid arthritis, who looked into her own future and contemplated the year 1949 with appalling hopelessness. At this time no published reports concerning the hormones had been made. His final remarks are as follows:

> This and other grim "side effects" of this vicious disease have, for years, disturbed me more than any of the side effects from cortisone or ACTH which I have seen develop in the past three years. As important as the latter are (and, rarely, they too can be tragic) they are the price of constructive effort rather than the price of despair, neglect or therapeutic nihilism.
>
> Thus it is my opinion that progressive rheumatoid arthritis involves distresses and fearsome hazards which fully justify calculated risks. The new hazard of mild hypercortisonism is being accepted by many physicians and patients in an attempt to provide a more bearable life for the miserable victims of this disease. Further experience and discoveries will reduce this hazard to the status of those already considered generally acceptable, such as cholecystectomy, hysterectomy; the automobile, airplane, slippery bathtub, electric stove; tobacco or alcohol.

I agree with Hench's remarks, because if the disease is not modified by other means we must assume a calculated risk.

Causes of Unsatisfactory Results.

1. Poor selection of cases may be a cause of unsatisfactory results. Disappointing cases are those in which there is little inflammatory activity, but in which there is extensive joint destruction. Pain on movement may be traumatic, the result of mechanical articular derangement, and not of inflammatory origin. Our experience has shown that success depends on the absence of gross joint deformity, and on the presence of early or active inflammation with little or no structural damage.

2. Failure may be due to too rapid tapering-off of dosage when the patient is doing well. I have found this happening in my early cases. Excessive activity of some patients when the symptoms of pain *et cetera* are suppressed by cortisone may produce excessive trauma and an unsatisfactory result.

3. Insufficient dosage may be a cause of failure. I have seen three patients who had received initially only 25 milligrammes of "Cortone" daily, but whose condition was improved when dosage increased. Another "failure" received ten milligrammes of ACTH every fourth day only.

4. Cortisone administered intramuscularly may form a "lake" at the site of injection and is slow in absorption; therefore the oral administration in small divided doses, as small as 12·5 milligrammes twice a day, has been more satisfactory.

Relapses and Remissions After Cessation of Cortisone Therapy.

The following results were obtained in the 65 cases: slow progressive relapse to pre-cortisone level, 18 cases; quicker relapse to pre-cortisone level, six cases; severe relapse (rebound attack)—condition of joints worse than before treatment, two cases; short post-hormonal flare of two to three weeks, then a prolonged secondary remission up to several months, 10 cases; prolonged remission (from three months up to two years), 29 cases.

It would appear that response to cortisone by rheumatoid arthritis subjects falls more or less into the following three main groups: Group I: patients whose symptoms are controlled by low (safe) maintenance doses, combined with physical therapy; Group II: patients whose maintenance dose requires adjustments every few days—suppression of symptoms is generally satisfactory; Group III: patients in whom control of symptoms is not possible in any dosage, owing to unknown factors such as poor utilization of cortisone or ACTH.

Discussion.

Should cortisone and ACTH be regarded as therapeutic agents for rheumatoid arthritis? It appears that there is no general agreement by physicians either in the United States of America or in Australia as to a practical form of treatment. Some hold that these hormones are for clinical investigators only, others that they are good remedies, and again they have been described as "very bad medicine".

Careful observations show that the successful use of cortisone and ACTH is achieved only by proper selection of cases, and thereafter by careful management. They are not agents for busy practitioners who can give only a very few minutes daily to a rheumatoid sufferer.

Programme III.

Programme III consists of chrysotherapy.

In my selection of cases all patients were given calcium aurothiomalate ("Auro-Calcium", Crookes Laboratories, London). In the past two years 857 patients were given

one to four courses of the gold compound, and in this series 12 patients (less than 2%) developed toxic reactions, which were controlled by stopping the injections. This is worthy of note.

It was found that small doses from 10 to 25 milligrammes a week for a longer period were more successful than a shorter course with larger doses.

Treatments of Choice.

Programme I is the treatment of choice for early or mild rheumatoid arthritis with little progression. It should be used with a full physiotherapy course for a prolonged period. If this combination fails to produce satisfactory results, a combination of programmes I and II—all general measures *plus* the administration of cortisone or ACTH—may be used.

A combination of programmes I and III—general measures *plus* gold therapy—may be used for patients intolerant to cortisone, or for reasons of cost.

The most successful treatment for prolonged remission in severe arthritis has been achieved by a combination of all three programmes. This has been effected by administering cortisone or ACTH at first alone, and then as soon as a remission is obtained by substituting one or both of the other programmes.

Still's Disease.

Two patients with Still's disease have been treated, with most satisfactory results. Another patient commenced treatment, but passed out of my care.

M.N., a male patient, had a sudden and severe onset five years ago at the age of thirteen years. He was first examined in November, 1950, then aged fifteen years, in a most advanced stage of arthritis. General examination showed him to be very emaciated; his height was four feet four inches and his weight 63 pounds. Both hip and knee joints were almost completely ankylosed, and the legs were fixed in a position of adduction, the left knee in 25° of flexion. The boy could scarcely feed himself and was in constant pain. The most significant special investigations showed that his hæmoglobin value was 65%, and the erythrocyte sedimentation rate was 40 millimetres for the first hour (Westergren).

In November, 1950, the cost of cortisone was prohibitive for his family, and Merck and Company Inc., New Jersey, U.S.A., generously made available free of cost five grammes of "Cortone Acetate", and treatment commenced.

This boy gained prompt and lasting freedom from pain, and movements of knee and hip joints were evident after four days' treatment. Progress continued, and with the help of Dr. John Jens, the limbs were gradually straightened over a period of eight months. During this period the disease was completely suppressed, and the patient grew 18 inches in twelve months, as well as gaining 28 pounds' weight. After fifteen months he began to walk about with the aid of calipers, which afterwards were discarded. A residual disability of one and a half inches of shortening of the left leg, due to atrophy of the head of the femur, has been greatly relieved by an osteotomy performed by Dr. Jens. The result has been highly satisfactory. Cortisone has not been given for over ten months now, and the patient has remained well. It appears that the arthritis has burnt out.

The other patient, J.O., aged sixteen years, has been under treatment now for five months, and is showing a favourable response to treatment.

Conclusion.

The conclusions to be drawn from the foregoing group of cases are that with some attempt to group types according to the severity of the arthritis, and with the cooperation of orthopædic surgery, greater and more lasting remissions can be attained. This survey does not include subjects suffering from osteoarthritis or from ankylosing spondylitis.

Summary.

Three programmes of treatment of rheumatoid arthritis are described: Programme I, the treatment of choice in mild or early cases; Programme II, the treatment in acute and more severe cases; Programme III, gold therapy.

Acknowledgements.

I gratefully acknowledge the practical help given by Dr. J. M. Carlisle, medical director, Merck and Company, Rahway, New Jersey, U.S.A. To Dr. John Jens, for his advice and cooperation in the surgical procedures in the treatment of Still's disease, the successful outcome in the case quoted is largely due. Special thanks are due to my masseur, Mr. R. Kemp, for devising various exercises.

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Reviews.

Famine Disease in German Concentration Camps, Complications and Sequels: With Special Reference to Tuberculosis, Mental Disorders and Social Consequences. By Per Helweg-Larsen, Henrik Hoffmeyer, Jørgen Kieler, Elgil Hess Thaysen, Jørn Hess Thaysen, Paul Thygesen and Munke Hertel Wulff; 1952. Copenhagen: Ejnar Munksgaard. 9½" × 7", pp. 460, with 25 text figures. Price: Dan. Kr. 35.00.

IN the Minnesota experiment reviewed in these columns in December, 1950, a detailed account was given of the effects of protracted semi-starvation on a number of young men. It must, however, be remembered that the subjects of the experiment submitted voluntarily to the restricted diet; they were under the control of kindly doctors and technicians and not capricious and brutally sadistic gaolers; if danger to body or mind was threatened the experiment, as far as any one of them was concerned, could be stopped; they were not harassed by the uncertainty of their home conditions nor of their fate whether they would somehow survive or be sent to the gas chamber; they were not given manual work to do beyond their powers; they were not flogged or otherwise tortured. Many clinical descriptions of survivors of German camps and prisons have been published, but unquestionably the best is a special supplement to *Acta medica scandinavica* which deals with 1282 Danes who survived varying periods of internment in German concentration camps during World War II. Here we have the real thing; not a laboratory experiment but an examination, necessarily mostly clinical, of the wrecks of humanity who escaped death in what is perhaps the most horrible instance of mass malignity in history. "Millions were killed by direct extermination, but millions more died of famine disease and its complications. Without taking direct murder into account the average duration of survival in these camps was little over six months." It would be a serious mistake to underrate the importance of the Minnesota experiment and a warm tribute to its value is paid in this volume—"the impressive and magnificent achievements of the Minnesota experiment". In this American research each organ and each function were examined by every technique and instrument of precision known to medical science and by every expert in clinical as well as laboratory inquiry. In the Danish report now presented the clinical perforce preponderates. There are a few other differences—for example, the psychiatric picture in the American inquiry reflects essentially the condition during starvation, the Danish two years after. Though the narration is presented with scientific restraint the story is none the less harrowing and "man's inhumanity to man" is only too vividly pictured. The Minnesota experiment never went as far as the grade called by the Danish investigators the "Mussulman"; this is a degree of emaciation in its terminal phase and is pathetic in the extreme. Amongst the many points of interest the following may be mentioned. "In the matter of brutal and ruthless treatment of the prisoners the Japanese camps fell little short of the German ones", but the Japanese gaolers gave a diet of higher caloric value to the prisoners: the result of this was that avitaminosis played a far greater role amongst Japanese prisoners than amongst German for the simple reason that a major function of vitamins is in the metabolism of food; the Japanese prisoners had some food to metabolize, but the German had little. The vicious circle of famine and disease is well brought out; famine

induces disease and disease accentuates the effects of famine. Tuberculosis, as might be expected, was the great agent of death before and after liberation. As has been noted elsewhere, sexual libido vanished during under-feeding and in many cases an impairment of the reproductive function persisted arising from irrecoverable degeneration of testicular tissue. With the Minnesota experiment and this Danish investigation medical science has now the full story of the results of under-nourishment and the means, not at all simple, whereby the unfortunate victim can be nursed back into something approaching normality in body and mind. This report is written not only in syntactically correct but in fluent and graceful English; the very few errors in spelling can be put down to the foreign printer and not to the author or authors.

Surgery of the Eye. By Meyer Wiener, M.D., and Harold G. Scheie, M.D., D.Sc., F.A.C.S.; Third Revised Edition; 1952. New York: Grune and Stratton, Incorporated. 10½" × 7", pp. 460, with 447 illustrations. Price: $15.00.

THE third edition of this book carries on the tradition of the two previous editions in its profuse illustrations of various ocular operative techniques. This liberal illustration of methods described is to be commended and is a real help to the reader; it is, however, a necessary help, for in common with many of the publications of our distinguished American colleagues, the written text is often difficult to follow and not seldom ambiguous. This is partly due to the employment of terms and phrases peculiar to the medical profession of America.

This third edition of Wiener's work also suffers (as did the second edition) from the fault that its virtues were limited by the boundaries of one man's methods. Also, there is evidence that Dr. Wiener is becoming rather set in his ideas, as we all tend to do as the years of our own experience accumulate. Certainly, some modern concepts are put forward, notably in regard to the surgery of ptosis and the surgery of the oblique muscles of the ocular globe; retinal detachment, also, is dealt with in a forthright and emphatic manner. Indeed, where the author puts his own views and those of modern surgical investigators, this is a good book and worthy of all ophthalmic men to be received. But when he trots out old text-book routines, he becomes banal and boring. To take a striking instance, six pages are devoted to the old, traditional, outmoded McReynold's technique of transplantation of pterygium. The whole conception of the pathology and surgical treatment of pterygium was revolutionized in 1948 by two men, working independently, one an Egyptian and the other an Australian. Of the six pages on this subject in Wiener's book, five lines are given (and they are inaccurate in their description of the technique) to the newer and now largely accepted method.

The book, however, is worth looking at for the illustrations alone, although the multiplicity of sutures reminds one of one's boyhood tangled fishing days.

Infectious Mononucleosis. By Sidney Leibowitz, M.D.; 1953. New York: Grune and Stratton. 9" × 6", pp. 172, with three text figures. Price: $4.75.

THE foreword to this excellent monograph points out that the clinical and hæmatological criteria for the diagnosis of infectious mononucleosis were established in the years 1920-1930. Since that time the literature has been devoted to refinements of diagnosis and to a study of the complications of the disease. This monograph critically reviews the symptomatology, clinical signs and hæmatological findings.

The section devoted to ætiology is brief, but there is an interesting discussion of such debatable aspects as the chronic form of the disease, reinfection and exacerbation. The author reviews and evaluates the literature and bases his conclusions on his own experiences. The discussion of serological findings is full and will be of value to the reader, whether he is student, practitioner or pathologist.

One of the most pertinent features of the book is the assessment of complications and the frequency of their occurrence. Modern literature tends to stress their occurrence perhaps too much, to the detriment of an appreciation of their actual significance. The author, in this regard, assesses the occurrence of hepatic involvement, and devotes a good deal of discussion to its similarity to infectious hepatitis; he also deals with hepatic biopsy findings and liver function tests. He points out that asthenia, the commonest complication, is probably due to hepatic involvement.

The section on treatment is disappointing in that no conclusive statements are made about the value of such antibiotics as aureomycin and "Chloromycetin". The author points out that the literature is equivocal in regard to the first and too minimal in regard to the latter to justify any definite conclusions. Generally in management, liver involvement must be assessed critically in all cases.

The monograph comprises 160 pages, is well set out and tabulated. It will prove to be of assistance to the physician, general practitioner and student, but will interest especially the general practitioner.

Office Psychiatry: The Management of the Emotionally and Mentally Disturbed Patient. By Louis G. Moench, M.D.; 1952. Chicago: The Year Book Publishers. 8½" × 6", pp. 310, with 81 illustrations. Price: $6.00.

"PSYCHIATRY WITHOUT TEARS" might well be the alternative title of this book, the aim of which is to discuss the management in the consulting room by the general practitioner of the emotionally and mentally disturbed patient. It deals with the various aspects of psychiatry in the orthodox order—the development of personality, psychosomatic medicine, the neuroses, the psychoses, psychotherapy and physical treatments. These are all discussed with a minimum of psychiatric jargon, and although somewhat oversimplified, the text gives the general practitioner an easily readable approach to these problems that so often receive scant attention.

The best chapters in the book deal with personality development and adolescence; the latter problem especially is approached with complete and sympathetic understanding. The chapters on psychotherapy rightly stress the desirability of a non-directive approach—"advice is overly used in psychotherapy".

Electric therapy is unfortunately referred to as "shock" treatment—a misnomer which has greatly increased the trepidation with which the average patient approaches this treatment. More emphasis might have been laid upon the fact that this treatment is most undesirable as a consulting room procedure—"anyone who undertakes it outside the hospital has a serious obligation to avoid its abuse".

This book, while providing a pleasant approach to the subject with its amusing cartoons, unfortunately will tend to add to the levity with which this subject is approached. Humour about psychiatric patients is only too frequently found, and it is felt that such "funnies" are out of place in a medical text-book. Fortunately the text provides a sympathetic approach to the whole subject, and although very American in atmosphere, most of the problems are translatable to the Australian scene.

Recent Advances in Obstetrics and Gynæcology. By Aleck W. Bourne, M.A., M.B., B.Ch. (Cambridge), F.R.C.S. (England), F.R.C.O.G., and Leslie H. Williams, M.D., M.S. (London), F.R.C.S. (England), F.R.C.O.G.; Eighth Edition; 1953. London: J. and A. Churchill, Limited. 8½" × 5½", pp. 348, with 92 illustrations. Price: 27s. 6d.

IN this edition of "Recent Advances in Obstetrics and Gynæcology" some chapters have been brought up to date, notably those on cancer of the cervix and erythroblastosis. New chapters on hypertension and pregnancy, functional uterine hæmorrhage and intravascular thrombosis have been added, in each case the high standard set by previous editions being maintained. The radiological study of the obstetric pelvis, fœtal-pelvic proportions and the placental site has been adequately dealt with by E. Rohn Williams.

The first chapter deals with the nutrition of the pregnant woman. Modern research seems to swing away from the all-importance of "special diets", and it is pointed out that in any civilized peace-time community the pregnant woman has an adequate diet with the possible need of some added iron.

The danger of comparing animal experiments with what occurs in human patients is stressed, and the condition of a premature infant should not be judged mainly by its birth weight.

In former editions of this book suggested additions to a pregnancy diet were stressed as important, but the writers have now come to the conclusion that they were mainly superfluous. On the other hand, in writing of lactation they point out that it is well to buy the essential vitamins and other "factors" from the dairy and greengrocer rather than from the chemist's shop.

A discussion on weight increase in pregnancy rightly stresses the fact that it is not the amount of the total gain but a sudden and rapid increment which acts as a warning of impending toxæmia—especially if linked with a rise of the diastolic pressure above 90 millimetres of mercury.

A well-reasoned chapter on erythroblastosis brings the reader up to date on this subject and attempts to explain such anomalies as the occurrence of the disease in children of Rh+ mothers and its greater frequency among children of compatible A B O blood group parents. Cæsarean section as treatment is rightly relegated to the scrapheap.

Perhaps one of the best sections in the book is devoted to hypertension and pregnancy.

Modern theories of the cause of toxæmia are summarized and a good deal of credibility is given to ischæmia of the placenta as producing a "toxin". Quite rightly, essential hypertension is stated not to be an indication for termination of pregnancy; but there is some departure from the usually accepted ideas of this country when it is maintained that the duration of toxæmia prior to induction of labour has no influence on the development of future hypertension.

In an excellent discussion of the diagnosis and treatment of cancer of the *cervix uteri* it is interesting to note the modern trend in treatment towards radical surgery as a substitute for, or an addition to, irradiation. In the sixth edition of this book the authors stated that radium and deep X-ray therapy had superseded surgery in this disease.

There is a particularly well-balanced and conservative review of stress incontinence with an evaluation of the modern methods of curing this distressing symptom. But we must strongly dissent from the authors when they lay blame for its causation on "that objectionable practice lateral or postero-lateral episiotomy".

The concluding chapter of the work deals with X-ray therapy in gynæcology.

Readers in Australia will be gratified with the large amount of credit given to the Sydney school—to Schlink *et alii* for their work in surgery *plus* radium for cancer of the *cervix uteri*, and to Cummine and Lyons for their conception of thrombosis and embolism.

This eighth edition more than lives up to the preceding ones and can be most enthusiastically recommended, especially for advanced post-graduate and specialist study.

Polyglot Medical Questionnaire: In Twelve Languages with Digital System of Communication. By S. Chalmers Parry, M.A. (Cantab.), M.R.C.S., L.R.C.P., D.P.H., with a foreword by M. T. Morgan, C.M.G., M.D., D.P.H.; 1953. London: H. K. Lewis and Company, Limited. 5" × 7½", pp. 62. Price: 28s. 6d.

IN the foreword to this useful little book we are told that "Dr. Chalmers Parry has spent many years in devising systems . . . whereby the barrier of language can be easily and rapidly surmounted". It is obvious that his time has been well spent. A most ingenious method is used in this *questionnaire*, what is described as the "digital" method, by which the use of the spoken word becomes entirely unnecessary. All that is required is that the examining doctor and the patient shall each be able to read his own language. The medical text consists of a series of words and phrases, numbered from 1 to 191, in twelve languages—English, German, Dutch, Norwegian, Swedish, Danish, French, Spanish, Italian, Portuguese, Russian and Polish. By selecting the appropriate phrases and words in the columns of his own language, and pointing to the same numbers in the columns of his patient's language, the doctor should be able to take an adequate (if not grammatically impeccable) history. It is suggested that the *questionnaire* will be useful for the medical and nursing staffs of general and isolation hospitals at home and abroad, and for ships' surgeons, port health officers, aerodrome medical officers *et cetera*, as well as for immigration authorities, consulates, shipping agents and masters of vessels not carrying a doctor. We think that it will.

Influenza: And Other Virus Infections of the Respiratory Tract. By C. H. Stuart-Harris, M.D., F.R.C.P., with a foreword by C. H. Andrewes, M.D., F.R.C.P., F.R.S.; 1953. London: Edward Arnold and Company. 9" × 6", pp. 242, with 98 illustrations. Price: 30s.

PROFESSOR STUART-HARRIS, who is most expertly qualified to perform the task, presents the facts on the clinical, microbiological and pathological aspects of influenza, the "febrile catarrhs", the bacterial pneumonias, "Q" fever and human infections with psittacosis-ornithosis viruses. Although the book is not overburdened with technical data, enough information is presented to acquaint the reader with the vast extension of knowledge on the influenza virus which has occurred in the last decade, and Australian readers will derive considerable satisfaction from the acknowledgement the author makes to the contributions to this important theoretical field by Burnet and his colleagues.

The author makes clear the necessity for further research on the "febrile catarrhs"—the respiratory infections which can be clinically distinguished from the common cold, but which are non-influenzal in ætiology. Despite their economic significance in the community, present information regarding ætiology is meagre.

Professor Stuart-Harris's unique experience with influenza enables him to treat the subject of its epidemiology authoritatively and in a stimulating manner. What happens to the influenza virus between epidemics is a question of perennial interest, and the author indicates that it may lie dormant during the summer months in a human or non-human host, or may be literally bandied about in the wake of the winter sun back and forth across the equator. The data he presents on the limitations and potentialities of influenza vaccination are of considerable interest and importance at the present time.

To practising doctors the author's closing remarks on antibiotic therapy in respiratory infections in general are wise counsel. Blunderbuss therapy for benign catarrh is deplored; and the poor prognosis in elderly patients with pneumonia complicating influenza is stressed as an example of the relative failure of antibiotics.

This important monograph is of convenient size and has the added merit of being of value to the specialist worker, the family doctor and the student.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"Experimental Studies in Psychiatric Art", by E. Cunningham Dax, B.Sc., M.B., B.S., D.P.M., with a foreword by Steuart Wilson; 1953. London: Faber and Faber, Limited. 9" × 6", pp. 100, with 51 illustrations. Price: 18s.

Deals with an experiment in the use of painting and music in the treatment of mental illness.

"Pathology in Surgery", by Edwin F. Hirsch, Ph.D., M.D.; 1953. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 6½", pp. 492, with 388 illustrations. Price: $10.00.

Intended for pathologists, surgeons and surgical "residents" of the various specialties in hospitals and medical schools and for medical students with some training in general pathology.

"Aids to Gynæcology", by W. R. Winterton, M.A., M.B., B.Ch., F.R.C.S., F.R.C.O.G.; Eleventh Edition; 1953. London: Baillière, Tindall and Cox. 6¼" × 4", pp. 204, with 15 illustrations. Price: 6s.

One of the well-known Students' Aids Series.

"Operative Gynecology", by Richard W. Te Linde; Second Edition; 1953. Philadelphia: J. B. Lippincott Company. Sydney: Angus and Robertson, Limited. 10" × 7", pp. 926, with 409 illustrations and seven plates in colour. Price: £10 15s.

An attempt to bring the subject of operative gynæcology up to date and to make recent information available in a single volume.

"An Atlas of Skull Roentgenograms", by Bernard S. Epstein, M.D., and Leo M. Davidoff, M.D.; 1953. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. 10½" × 7", pp. 416, with 603 illustrations. Price: £8 1s. 3d.

Emphasis has been placed on plain skull röntgenography.

"Hormonal and Neurogenic Cardiovascular Disorders: Endocrine and Neuro-Endocrine Factors in Pathogenesis and Treatment", by Wilhelm Raab, M.D., F.A.C.P., F.A.C.C., F.C.C.P.; 1953. Baltimore: The Williams and Wilkins Company. Sydney: Angus and Robertson, Limited. 9" × 6½", pp. 744, with 86 illustrations. Price: £8 1s. 3d.

Deals with the neuro-endocrine and endocrine influence in cardio-vascular disorders and the need for its recognition.

"Survey of Clinical Pediatrics", by Lawrence B. Slobody, M.D.; 1952. New York: McGraw-Hill Book Company, Incorporated. 9½" × 6½", pp. 480. Price: $7.50.

Intended for the student and practitioner.

The Medical Journal of Australia

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All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

THE PRACTITIONER'S DOUBLE DEBT.

THE practitioner of medicine is one of the most richly endowed of mortals. He is endowed by tradition, by his training which gives him a direct entrée to the gates of knowledge and understanding, and by the inevitable closeness of his association with men and women which gives him opportunities of service possessed by no other professional man, with the possible exception of the priest. His tradition carries him back to the medicine of ancient Egypt, the oldest of which we have historical knowledge; from Egypt his heritage comes down through Babylon and Greece and through the darkness of the Middle Ages to the present day. All through the ages there have been ethical codes and penalties have been imposed for transgression; the obligations of medical practitioners have not varied. When the student enters the medical school to start his course he becomes heir to the endowment of the practitioner and on graduation he enters into his inheritance. He may take as his own all the knowledge of his predecessors; if he is wise he will study their history, for he has to assume obligations as well as equip himself with knowledge. In other words, he has received benefits and he has to pay corresponding debts. These may be considered under two headings—first of all to persons, and secondly to medicine. Of these the first is much more generally recognized that the second.

The first is said to be to persons rather than to patients, because the debt is not only to those who are sick and need treatment, but also to those who are well, that they may remain well and not require treatment. Even the departmental medical officer, sitting in his office, who often seems remote from persons, cannot escape the personal debt. Fortunately he seldom tries to. The private practitioner who wishes to pay his proper debt to the persons among whom he practises must prepare himself in several ways. First of all he must remain a student. This means that he must make post-graduate study part of his everyday life. We all know exactly what this implies. It does not mean that he is to be a book-worm and nothing else—systematic reading, attendance at post-graduate lectures and at meetings of the British Medical Association and other scientific societies, and discussions among his fellow practitioners all have a place. If he were to be a whole-time book-worm, he would neglect the second kind of preparation, which is to keep himself fit—in body and in mind—to render the service required of him. We all know what this means, too—the "daily dozen", the round of golf, the cult of the lawn mower, the call of the surf and so on, not forgetting systematic reading or browsing in general literature and devotion to the arts. If the practitioner is to make others whole, he must be a whole man himself. But this keeping fit business must not be used to cover up absence from the consulting room or to justify unwillingness to meet the legitimate requirements of his patients. It can never explain that unworthy arrangement known as a "lock-up" practice. The third kind of preparation for the payment of debt to persons is to be personally available to them when they are in need of attention. The fact that a doctor puts up his plate outside his consulting room is an indication to the public that he is willing to be consulted by sick persons and to do what he can for them in the matter of treatment. He generally has what are known as consulting hours, and the implication is that he will be found in his rooms at those hours unless he has been called to a patient who is in need of urgent and immediate attention. The implication is also that he will be willing to attend patients whenever they are in need of his help. It can be stated beyond any doubt that when a general practitioner knows that he will be absent from his practice for any considerable period of time for any reason whatever, he should make arrangements for another practitioner in the vicinity to be "on call" for him. The recent complaint of a "columnist" in one of the daily newspapers that a person taken ill suddenly at the week-end could not secure the attendance of a doctor even after an enormous number of telephone calls, is not an exception. Many doctors do work on a roster at week-ends, but sometimes it seems to be impossible for the public to find out who is on duty. No one expects a doctor to be on duty twenty-four hours a day for seven days a week, but everyone has a right to be able to secure medical help in an emergency at any hour of the day or night. This is a matter to which organized medical bodies such as local medical associations should pay urgent attention. Disease indeed "knows no holiday"; and medical practitioners will never enjoy a forty-hour week, or any other week of stated hours unless they entirely lose their freedom.

So much for the practitioner's debt to persons; his debt to medicine is just as clamant. This subject was recently discussed in an address by William A. Barrett[1] at a meeting of the North-Eastern Section of the American Urological Association aboard the S.S. *Ocean Monarch* in October, 1952, on a cruise to Bermuda. Barrett points out that since the practitioner's education is never complete and since he draws heavily on the accumulated mass of knowledge as long as he practises, his indebtedness increases much more rapidly than his ability to make any

[1] *J.A.M.A., March 14, 1953.*

contribution to the total. For the sake of his patients "he must never stop borrowing, and, for his own sake as well as for that of the profession, he must never cease striving to repay". He sets out three ways in which the debt may be repaid. First each practitioner must continue the advancement of medical lore, must contribute to knowledge and to the maintenance of professional ideals. The second method is described in the Hippocratic Oath: "Teach them this art, if they shall wish to learn it, without fee or stipulation; and that by precept, lecture and every other mode of instruction." Barrett states that the maintenance of the traditionally high standards of the medical profession necessitates the training of younger men in the art and in the science, and that, even more, it necessitates in each member the development of personal sincerity, of stark honesty with himself, his colleagues and his patients, and of utmost integrity. This, of course, opens up large questions of post-graduate training which cannot be dealt with at the moment; it must suffice to state that an effort to teach others the significance of tradition, history and usage will be of the greatest benefit to the teacher. Barrett's third method of repayment is to strive to maintain "the most priceless privilege medicine has enjoyed throughout the ages, freedom". It should not be necessary to emphasize the value of professional freedom to readers of this journal in view of the medico-political happenings in the Federal sphere during the last few years. Medical practitioners in Australia have reason to value their freedom. This discussion on the debt of all practitioners to persons and to medicine itself, if it does nothing else, shows that freedom does not come for the asking—it has to be deserved and it has to be worked for.

Current Comment.

THE EQUIPMENT FOR THE CONQUEST OF MOUNT EVEREST.

THE success in the climbing of a mountain as high as Mount Everest depends on many things, not least being the nature of the equipment for the members of the climbing party. Each expedition has the advantage of the knowledge gained by all the previous attempts, and the expedition led by Sir John Hunt, two members of which achieved the summit, has added to this experience carefully planned laboratory and field tests of critical equipment such as tents, bedding, clothing, food and oxygen supply. In a special supplement to *The Times*, London, Sir John Hunt has given some details of the type of equipment used. The climatic problems of cold and high wind, progressively enhanced as they are by the rarified atmosphere, posed the greatest problems in the design of equipment, and the imperative need to reduce weights to a minimum did not make the task any easier. As regards clothing an outer suit of cotton windproof material consisting of smock and trousers, double lined with nylon and of a combined weight of about three and three-quarter pounds, was worn. This was mainly to give protection against wind. The smock had a hood with visor to provide protection from wind and snow. Differing colour of this outer garment was chosen to simplify identification of individuals. Inside this a two-piece suit of down, also with a hood, was worn. This was very light, while giving good protection against cold and reducing the number of woollen garments needed. Two types of boots were worn. The first, for use up to about 23,000 feet, was similar in design to the normal mountaineering boot, but had a double leather upper with fur between the layers and the leather was specially treated to resist freezing. For the final assault a special type of boot combining lightness with exceptional insulation against cold was designed. The uppers of these boots consisted of almost one inch of propal contained between a very thin glacé leather and an inner layer of impermeable cloth. The soles were made of special microcellular rubber, very light with excellent insulating properties. On the hands an outer gauntlet of windproof cotton enclosed either a down or woollen mitt. Next to the skin was a close-fitting silk glove which besides giving additional warmth to the hands inside the mitt, could be worn alone for short periods when it was necessary to perform some intricate task. The tents were of the normal type with sleeve entry which provided internal communication between tents, but were made of cotton and nylon woven together, proofed by a special process, and light, strong, windproof and waterproof. The sleeping bags had an inner and an outer bag of down in nylon fabric specially constructed so that there was no loss of insulation at the line of stitching between compartments. For food two types of composite ration, in current use by the British Army, were used, the so-called "Compo" ration made up in thirteen man-day packs, and the "snow" ration, a small two pound one man-day pack modified somewhat for use above the advanced base. The oxygen apparatus worked on the "open-circuit" principle, that is, oxygen was given from a bottle on the back and breathed out into the surrounding air. The design of the apparatus was greatly superior to what had been used before, giving oxygen for about five hours. Another type of apparatus was taken, but there is yet no indication as to whether it was used. In this the climber received 100% oxygen from the bottle and rebreathed a proportion of the expired air, thus increasing considerably the life of the storage bottle. That the equipment was adequate is evident from the success of the expedition and the apparent lack of any serious mishaps. The fabrics used in the clothing and tents were specially developed by the British textile industry for this expedition.

A STUDY OF HÆMOGLOBIN VALUES IN NEW SOUTH WALES.

"A STUDY OF HÆMOGLOBIN VALUES IN NEW SOUTH WALES", by R. J. Walsh, B. J. Arnold, H. O. Lancaster, M. A. Coote and H. Cotter, has been published as Number 5 in the special report series of the National Health and Medical Research Council. The work described in this report was performed in the laboratories of the New South Wales Red Cross Blood Transfusion Service. The authors, however, did not limit their investigation of hæmoglobin values to blood donors, but extended it to all classes of the community so as to present a valuable and comprehensive piece of work. In general principles, the survey follows closely that of the Medical Research Council of Great Britain published in 1945. It will be remembered that the object of the British survey was to obtain some evidence about the nutritional state of the people of Great Britain in the fourth year of the war. The aims of the present survey were twofold: firstly the determination of mean values for hæmoglobin, and secondly the detection of low values possibly associated with nutritional deficiencies in the entire population or in certain groups. In an introductory chapter, these aims are discussed briefly, together with various physiological considerations affecting erythropoiesis.

The second chapter of this monograph describes the making of a hæmoglobinometer. At the outset, it was necessary to procure an instrument which would allow accurate and rapid determinations of the hæmoglobin concentration of large numbers of blood samples and to calibrate it so that the results obtained would be comparable with those of other surveys. As the authors point out, dilution methods are subject to large errors, and colour-matching either by inspection or in a colorimeter introduces subjective errors on the part of the operators.

Photoelectric hæmoglobinometers are free from these disadvantages, but must be carefully constructed. After investigation of several commercially produced instruments, it was decided to construct a single-cell photoelectric hæmoglobinometer for the survey. The design and construction of this instrument are described in detail. The completed instrument was calibrated on the basis of blood iron content; this was done in two ways: first, by using results obtained by the National Physical Laboratory, Teddington, England; and secondly, by sending samples of blood to Brisbane, where their hæmoglobin contents were determined on Beckman spectrophotometers at the Queensland Institute of Medical Research and at the Department of Physiology, University of Queensland. Venous blood was used for all tests on adults, following an investigation of the variability of hæmoglobin values obtained from the finger and the ear. This follows the method used in the Medical Research Council survey, in which it is stated that "the best site for taking blood" (for hæmoglobin determinations) "is a vein, preferably that of a warm arm".

The following two chapters deal with the hæmoglobin values of adults in New South Wales. Samples of blood were taken from a group of blood donors selected at random, from volunteers examined at an exhibition arranged by the Sydney City Council in connexion with Health Week during October, 1949, from various industrial and other groups, from a very small group of donors in the inland cities of Goulburn and Cowra, and from the Newington and Lidcombe State Homes and Hospitals for the aged. In addition, by arrangement with the staff of The King George V Memorial Hospital for Mothers and Babies, 1043 hæmoglobin estimations were performed on women at various stages of pregnancy. The results are summarized as follows:

> The mean value of hæmoglobin in 1792 males was 15·71, and of 1230 females 13·89 grams. No significant difference was found between married and unmarried subjects of either sex. These values compare favourably with those of other countries, and the male value is higher than that found in the United Kingdom in 1943.
>
> There was a progressive decrease in the mean values of both sexes with increasing age, the decrease first becoming apparent in males between 60 and 69 years and in females between 70 and 79 years.
>
> The parity of married women did not influence the mean hæmoglobin values after the menopause, but inconstant and relatively small differences were observed in the younger age groups.
>
> The mean value in blood donors was not influenced by the amount of blood which had been removed.
>
> It was not possible to assess the slight differences encountered in different occupational groups because other factors, such as season, could not be eliminated. The mean value of comparable groups tested at different times of the year showed a lower level in summer than in winter.
>
> The hæmoglobin concentration of pregnant women showed a marked and progressive fall as pregnancy advances, but the values of 1043 women at different stages of pregnancy were almost identical with those observed in England. A slight increase in the mean values was found toward the end of pregnancy.

The progressive reduction of hæmoglobin values with increasing age in both sexes is an interesting finding, for which no satisfactory explanation has so far been found. As regards the hæmoglobin value in blood donors who had given a varying number of donations of blood, one feels that the present survey was somewhat limited in extent. Only 160 such donors were examined. The number of donations ranged from five to twenty or more. No evidence was found that repeated removal of blood reduced the mean hæmoglobin value. The authors compare this with the findings of Bryce and Jakobowicz (1943),[1] who showed that whilst there was no alteration in the mean values of males, those of female blood donors showed a progressive decrease with successive donations. The numbers examined by Bryce and Jakobowicz were far greater than those of the present series. They estimated the hæmoglobin value of a series of 1577 men and 3957 women who had given blood on two or more occasions at intervals of not less than three months, as well as of a series of 4110 men and 8956 women prior to the giving of blood.

With the instrument used in this survey, small physiological variations could be measured. The effects of exercise, ingestion of fluid, time of day and site of blood collection were the subject of experiments recorded in Chapter V. As mentioned above, it is recommended that venous blood should be used whenever possible; also that blood should not be obtained from the ear, and that if the finger is used it should not be cold, nor should it be heated by immersion in hot water.

In a study of hæmoglobin values of infants and children, volunteers were obtained from the following groups: 192 newly born infants, selected at random from the Royal Hospital for Women, Sydney; 1010 children attending baby health centres throughout the Sydney metropolitan area; 613 children attending day nurseries; 554 children attending kindergartens; 1529 children attending primary schools and 554 in secondary schools in the Sydney area. In addition 336 children were examined during Health Week. Previously reported changes with age were confirmed. It was found that hæmoglobin values in the Sydney area compare favourably with those obtained in Britain in 1943 and in general are higher than those found in Edinburgh and Aberdeen. For various reasons the values found in Sydney, like those in surveys elsewhere, are not necessarily to be regarded as optimal. The authors weigh the evidence, from their own and from other surveys, that a deficiency of iron may occur during the first year of life.

Hæmatocrit values and sedimentation rates, determined on a series of blood donors of both sexes ranging in age from eighteen to sixty-three years, are analysed. Wintrobe's hæmatocrit tubes were used for both determinations. The results of various alterations in technique are recorded. The ranges of sedimentation rates found for both sexes were considerably greater than those usually quoted. It is true that no attempt was made to exclude those suffering from mild infections; still, the subjects were all apparently well and the authors aver that the ranges encountered in their series are those likely to be seen in clinical medicine, and are such as to render the test of little value from a diagnostic point of view. Experienced clinicians will disagree with this statement. The sedimentation rate is not a specific test; it must be correlated with clinical and with other laboratory findings, but when used with intelligence and experience it can be a most useful piece of evidence.

Altogether this report is a thorough and valuable piece of work, recorded with precision and lucidity. To attempt to convey this impression, the authors' own words have been used freely in this abstract; but the whole report must be read and studied to be appreciated. The elaborate statistical analyses represent a great deal of hard work and careful thinking. The work provides an essential standard for clinical reference for the Sydney area, and a firm basis for future work in the fields of hæmatology and nutrition. It would be most interesting if the authors could extend their work to the population of the inland and outback areas of New South Wales.

[1] Bryce, Lucy M., and Jacobowicz, Rachel (1943), "The Hæmoglobin Value in Blood Donors", M. J. AUSTRALIA, 2: 329.

RECONSTRUCTION OF THE MALE URETHRA.

ONE of many small but significant features of the antibiotic age is a decline in the incidence of urethral stricture, the result no doubt of the effective treatment of acute gonorrhœa. According to John Swinney,[1] it has been estimated that no less than 66% of strictures take fifteen or more years to develop, only 5% appearing within five years of the initial lesion, so that we may expect the decrease in incidence from gonorrhœa to continue for some years, although the rise in incidence of acute non-specific urethritis may offset this to some extent. In any case, the treatment of such strictures as continue to occur

[1] *Brit. J. Urol.*, September, 1952.

presents considerable practical problems. The time-honoured treatment and, in general, as Swinney states, the most successful treatment of stricture of the urethra is intermittent dilatation with sounds. When this fails, however, various open operations have been tried, such as urethrotomy and excision. In the latter case, with end-to-end anastomosis, one would think that the results would be good, but this has not been Swinney's experience. There are some cases of stricture which have, up to the present, been most difficult to ameliorate by any means. These are: (*a*) the resilient type, (*b*) the gristly or hard type, and (*c*) the stricture behind which abscesses and fistulæ have formed. It is the problem of such difficulties that has impelled Swinney to search for a better method. He has been intrigued by the modern work on the fate of a flat strip of skin which is left buried under the subcutaneous fat. Davis and Traut in 1926 sutured whole-thickness skin grafts to the abdominal muscles of dogs and covered them with the deep fascia and skin. Rounded or square pieces of skin showed proliferation of the epithelium from the edges, so that finally a closed cystic cavity was formed, lined by epithelium. Oblong pieces of skin showed the same effect; in this case, long tubular cysts were formed. In 1949 Denis Browne, of London, used this principle in devising his operation for hypospadias. From the ectopic meatus forwards, he outlines a strip of skin to the end of the penis by parallel incisions and, after mobilizing the skin of the lateral parts of the penis and making a dorsal relieving incision, he sutures the medial edges of the mobilized skin over the outlined skin strip. The buried flat strip forms a tube, which is the new urethra. This procedure is very simple, and has proved highly successful. In 1950 Reed Nesbit and others, working on male rats at the University of Michigan, buried a strip of abdominal skin under the mobilized surrounding skin, and left each end continuous with the surface skin. A tube was duly formed. At two-day intervals, these tubes were examined microscopically. It was found that, after completion of epithelial growth, partial atrophy of the hair follicles and accessory structures began to take place. The calibre of the tubes remained comparatively satisfactory. A study of these facts led Swinney to the hope that this principle might be of use in the management of difficult cases of urethral obstruction. To ensure success, he lays down certain basic principles. The first is that all diseased tissue must be removed ruthlessly. This involves excision of all fibrosed urethra and *corpus spongiosum*, and all fistulæ and abscesses. The second principle is that the resultant raw area must be covered by skin, so that later on a strip may be outlined wide enough to form the new urethra. Instead of skin, if the roof of the urethra, left after excision of all fistulæ as well as the unhealthy floor of the canal, is wide enough for the purpose, it may be left in place and buried at once by closing the perineal tissues over it. It will be seen from this description that suprapubic cystostomy is the only possible method of urine deviation. Also, the cut ends of the urethra, both proximal and distal, must be sutured to the surface skin, and they must be left well open. The final stage of the reconstruction is as the earlier part of the procedure foreshadows. Approximation of the lateral skin flaps to bury the skin strip is effected with interrupted sutures, metal beads being clipped on in such wise as to hold the skin edges lightly in apposition. Swinney reports that the results have been so satisfactory in a short series of seven cases that the operation can be recommended. In one case it is to be noted that he actually did perform excision of a short stricture, anastomosing the flat strips of urethra, but he buried this strip at once by closing the perineum over it.

This work is of the greatest interest. To the Australian surgeon it will recall the important work of the late R. Hamilton Russell, of Melbourne. Passing reference is made by Swinney to Russell's work, but no details are given of it. Russell described his procedure in *The British Journal of Surgery*, Volume II, 1914-1915, at page 375. The article, which is freely illustrated, will repay study at this stage. His operation was based on the fact that the male urethra may be split from the membranous portion to the meatus or to a less extent with the assurance that no harm will result, and that no difficulty will be experienced in restoring its integrity, provided efficient drainage of the bladder is secured during healing. Russell excised the stricture and joined the ends of the split urethra so that a "riband" is formed. Those who are used to the Russell operation will not readily abandon it.

SALICYLIC ACID POISONING IN SKIN THERAPY.

POISONING by salicylic acid and its derivatives has not received a great deal of attention. Mild toxic effects are accepted as a necessary concomitant of adequate therapeutic dosage of salicylates in conditions such as rheumatic fever, and this may have obscured the fact that severe toxic effects and resultant death have been recorded. According to figures compiled by Gross and Greenberg, and quoted by E. P. Cawley, N. T. Peterson and C. E. Wheeler,[1] 526 accidental deaths from poisoning by all salicylates occurred in the United States during the ten-year period from 1933 to 1943. Figures for non-fatal poisoning are much more difficult to compile or even to guess, but it would be unwise to under-estimate them. Poisoning by salicylic acid absorbed from the skin during topical therapy is very rare, if we may judge by the recording of cases in medical literature, but this is not necessarily a reliable criterion. Cowley, Peterson and Wheeler report two cases occurring in the same year at the University of Virginia Hospital. Both the patients were children undergoing treatment for severe ichthyosis. One child had the whole skin anointed frequently with 3% of salicylic acid in equal parts of cold cream and "Aquaphor". After forty-eight hours she became emotionally upset, complained of abdominal pain and vomited. Later she had delusions of insects crawling on her bed and became semistuporose. The inunction was stopped when the diagnosis of acute salicylate poisoning was made, and recovery commenced almost immediately, being complete in seventy-two hours. The comment is made that the relatively large cutaneous surface in respect to body weight (the child was poorly developed and greatly underweight), the rubbing used in applying the ointment, the concentration of salicylic acid in the ointment, daily tub baths and the skin disorder itself were probable factors in facilitating absorption of significant amounts of salicylic acid in this instance. In the second case, a six-year-old boy of normal development showed no untoward reaction to application of salicylic acid ointment in strengths of from 2% to 5%; but generalized inunction with ointment of 10% strength (the result of a misinterpreted order) brought severe symptoms of vomiting, abdominal pain, emotional disturbance and increased respiratory rate. With cessation of use of the ointment and intravenous administration in succession of isotonic sodium chloride solution, 5% glucose in water and sodium lactate solution, symptoms subsided to disappear in forty-eight hours.

Cowley, Peterson and Wheeler describe the pharmacology, clinical manifestations and treatment of salicylate poisoning in general terms. They point out that plasma salicylate levels depend to a large extent on the dosage and mode of administration of the drug and on the excretory capacity of the kidney. Most of the drug is excreted in the urine. With a constant dosage of salicylates, the plasma level increases with a decrease in the pH of the urine and *vice versa*. Renal impairment enhances the possibility of salicylate poisoning. Several observers have demonstrated that salicylates applied to normal skin can subsequently be found in the blood and urine. The rate and degree of absorption of salicylic acid from the skin depend on several factors, including the concentration of the drug, the vehicle employed, the extent and duration of its application, cutaneous defects, presence or absence of hyperæmia, massage and bodily temperature. The clinical manifestations of salicylate poisoning result from various combinations of hæmorrhagic and degenerative changes in brain, kidney and skin, as well as central stimulation of the respiratory centre, toxic effects on the acoustic and vestibular organs, and alterations in the

[1] *J.A.M.A.*, January 31, 1953.

electrolytic balance. Prothrombinopenia, which is associated with an increase in prothrombin, bleeding and clotting times, in addition to increased capillary fragility, accounts in part for the hæmorrhagic tendency. Effects produced are dizziness, difficulty in hearing, tinnitus, mental confusion, sweating, thirst, nausea, vomiting and diarrhœa. Ecchymotic and petechial lesions occur on the skin. Treatment is symptomatic in most cases, and unless manifestations are unusually severe discontinuance of the drug will suffice. In severe cases dehydration must be combated by administration of fluids. Vitamin *K* is of value for patients with an increased prothrombin time, or clinical evidence of hæmorrhage. Available evidence suggests that sodium bicarbonate may be given orally as a prophylactic measure against the occurrence of toxic salicylate blood levels when salicylic acid must be applied over large areas of skin. Certainly the possibility of salicylate poisoning should be kept in mind. In milder forms it may be more common than the records in the literature suggest.

NURSES AND ANTIBIOTIC SENSITIVITY.

AN American report on sensitization of nursing staff to streptomycin, the result of contact with the drug in their nursing duties, was mentioned in these columns on May 29, 1948. However, there have been comparatively few references in medical literature to sensitivity to streptomycin or other antibiotics acquired in this way, and it might be inferred that the condition is rare. This is not so, according to the findings of a survey published in a recent memorandum from the Ministry of Health in Great Britain.[1] The survey, which covered 70 local health authorities and 76 chest hospitals and sanatoria, revealed 256 cases in which members of the staff had become sensitive to an antibiotic as a result of handling it in their work. The population at risk could not be learned for the whole survey; but where this population was known, the incidence of sensitization was 4·3% for the local health authorities, 1·8% for the hospitals and 3·5% for the two groups combined; in other words, between 1% and 5% of nurses using antibiotics became sensitized to them. The survey data do not indicate that previous allergic manifestations are of much importance in susceptibility, but this impression may be misleading; certainly, a severe degree of sensitization to one antibiotic is often associated with sensitization to others. Streptomycin and penicillin were the principal sensitizing agents. In a few severe cases the reaction included angioneurotic œdema, but in all cases the skin was involved; the main sites were the hands, arms, face, eyes and neck. The importance of local contact is thus obvious, and it appears to be of two kinds: first, direct contact of the liquid or powder with the skin, and second, contact with air-borne particles, either droplets or dust. The first of these, it is pointed out, may occur during the dispensing or preparation of injections, or result from breaking containers or spilling their contents, from handling contaminated swabs or syringes, or from touching the patient's skin; it should be entirely preventible by using careful technique and by wearing gloves. The second type of contact may occur when air (with a fine spray of the solution) is expelled from the syringe as a preliminary to giving the injection, when powdered preparations are handled, and possibly when steam escapes from a sterilizer. The commonest of these circumstances is the expulsion of the air from the syringe, a routine practice carried out automatically by most people in a fairly uniform fashion with the syringe held vertically at eye level. Two precautions should overcome most of the difficulties associated with injections: first, the air should be expelled before the needle is removed from the bottle or phial from which the solution has been withdrawn; second, the same needle should be used for both withdrawal and injection. Details of the appropriate technique and further comments on these precautions will be found in a Health Ministry Circular quoted in our "London Letter" in the issue of this journal for July 18, 1953.

Another important aspect of the problem is the need for early and accurate diagnosis of sensitivity. Early diagnosis with appropriate action will often forestall serious or permanent sensitization and save much trouble. The memorandum mentions swelling of the eyelids as a very early sign, especially of developing streptomycin sensitization. Accurate diagnosis is important, as the antibiotics can be unfairly blamed when the guilty agent is something quite different—a drug, cosmetic or plant encountered only coincidently, or an antiseptic or ancillary agent (such as procaine) associated with the antibiotic or its administration. The memorandum points out that no nurse should be regarded as sensitized to antibiotics without conclusive evidence. This is not the superfluous comment that it might appear to be, as the survey provided many instances of the serious effects of a diagnosis of sensitization, ranging from the need for the nurse to be given work not involving contact with the antibiotic concerned to the actual abandonment of nursing as a career. Such effects matter considerably to the community as well as to the individual nurse. They can be largely prevented by early and accurate diagnosis of developing sensitivity, as well as by careful attention to the precautionary measures described in the Health Ministry's circular.

[1] *Lancet*, July 4, 1953.

HYDATID INFECTION IN CANADA.

IN his classical book on hydatid disease, H. R. Dew referred to the rarity of this infection in North America, but expressed the opinion that the increase in the number of sheep pastured and the inevitable increase in the number slaughtered for human consumption in abattoirs or on scattered farms would lead to a gradual increase in the number of cases of hydatid disease. That was written in 1927, but the general view, according to Max J. Miller,[1] is still that human hydatid cyst infection is rare in Canada and that any such infections found are of foreign origin. Only a few cases have not fitted in with this, although there was evidence that the disease was endemic in Canadian wild life. Then the Indian Health Service discovered that hydatid infections were showing up with relative frequency in the Indian population of north-west Canada, and in 1952 a survey was carried out. It appears that the tapeworm, *Echinococcus granulosus*, is well established in the carnivora of a huge area which stretches across northern Canada and dips down to include most of British Columbia and which supports a rich fauna of wild herbivora and carnivora. The wolves, in particular, seed much of this area with tapeworm eggs. The eggs are picked up by the herbivora, usually moose or caribou, in which the hydatid cysts develop. The cycle is completed when wolves kill and eat the flesh, including the infected lungs, of these hosts. Miller points out that this cycle of "sylvatic echinococcosis" has probably existed in Canada for centuries and is of little economic consequence. It is only when man infects his dogs by feeding them lungs of caribou or moose that he initiates a cycle in his immediate environment; in this he inevitably plays the role of intermediate host, in the fashion only too familiar in Australia in the past. Apparently accidental infection in this way happens often enough among the Indians to constitute an important medical problem, and its control is being considered. Miller points out that control of sylvatic echinococcosis is not feasible, so that the infected dog needs to be dealt with. Control measures should aim at protecting the dogs against acquiring infection and eradicating infection already present in dogs. The first of these involves education of Indian and Eskimo populations against feeding uncooked moose and caribou viscera to their dogs and is apparently thought to be practicable. For the second aim a programme of treating infected dogs with tæniacidal drugs is being considered—a task that sounds much more formidable.

[1] *Canad. M. A. J.*, May, 1953.

Abstracts from Medical Literature.

OPHTHALMOLOGY.

Neuromyelitis Optica.

GEORGE I. SCOTT (*Am. J. Ophth.*, June, 1952) reviews the literature and reports ten cases of *neuromyelitis optica*. He concludes that at the onset there is often a history of sore throat or febrile disturbance which precedes the visual loss or myelitis. Pain on movement of the eyes is also common. In most cases the visual loss was the major incident, and the patients in these cases have a better prognosis than those in which myelitis appears first. There is no sex preference, and the disease is commonest between thirty and fifty years of age. There is no relation between the severity of the optic neuritis and that of the myelitis. The fact that it is often preceded by a febrile illness, the acute and bilateral nature of the optic neuritis and the pain in the eyes place it in a different category from disseminated sclerosis. In addition euphoria, nystagmus and cranial nerve palsies do not occur in *neuromyelitis optica*, and the colloid gold test gives a negative result.

Operative Treatment of Congenital Ptosis.

G. I. SCOTT (*Brit. J. Ophth.*, July, 1952) advocates advancement and resection of the levator, modifying Blaskovic's original technique, as the operation of choice for congenital ptosis. He describes his technique in detail and reports the results of 25 consecutive operations. In 20 cases the result was perfect or nearly perfect. The author regards the state of the superior rectus as the main factor in governing the choice of operation. If the superior rectus is not paralysed, then resection and advancement of the levator are advocated. If, however, the superior rectus is paralysed, then, providing the frontalis is active, a *fascia lata* graft should be performed with the technique described by Lexer. However, if the frontalis is not active, then an under-correction of the ptosis should be performed by resection and advancement of the levator. It is recommended that the operation be performed on patients at three years of age or sooner in severe cases.

Ocular Cystinosis.

HARRIET G. GUILD *et alii* (*Am. J. Ophth.*, September, 1952) describe two cases of systemic cystinosis and draw attention to the ocular manifestations. They state that cystinosis is a rare disorder characterized by the universal deposition of cystine crystals throughout cells of the reticulo-endothelial system. Most cases are characterized by chronic acidosis and renal glycosuria as met with in Fanconi's syndrome. However, opinions differ on whether cystinosis and Fanconi's syndrome are different stages of the same disorder or whether they are merely related disturbances with a common denominator in the form of a congenital defect in amino acid metabolism. Examination of the corneæ in the two cases described showed only slight haziness; however, on slit lamp examination myriads of refractile rod-shaped and needle-shaped particles were seen in both corneæ. These were distributed uniformly throughout the corneæ of both eyes. As far as could be determined the irises, lenses and vitreous were normal and contained no crystals. Other corneal affections may be confused if slit lamp examination is omitted—for example, Hurler's syndrome, band-shaped keratitis and corneal changes seen in Still's disease.

The Pathology of Early Retrolental Fibroplasia.

ALGERNON REESE *et alii* (*Am. J. Ophth.*, October, 1952) describe the results of histological examination in 10 cases of retrolental fibroplasia together with examination of 458 eyes of stillborn infants or premature infants who died a few hours to a few days after birth. The authors were able to show that the process is at first confined to the nerve-fibre layer in the equatorial region. It begins as a diffuse thickening due to small nests of endothelial cells and an increase of the glial elements. The proliferated endothelial cells canalize and break through the internal limiting membrane. Hæmorrhages occur from newly formed vessels, and then organization, contracture and folding of the retina begin. Angiomatous tissue creeps between the retina and the hyaloid membrane of the vitreous and then invades the vitreous. Evidence of retrolental fibroplasia was found in two sets of eyes of stillborn children out of 458 eyes examined.

Peripheral Iridectomy in Narrow-Angle Glaucoma.

J. S. HAAS AND H. G. SCHEIE (*Tr. Am. Acad. Ophth. & Otolaryng.*, August, 1952) offer an explanation for the mechanism of peripheral iridectomy in narrow-angle glaucoma. They believe that peripheral iridectomy functions by eliminating the mechanism responsible for so-called physiological *iris bombé* with resultant deepening of the anterior chamber. They further are of the opinion that peripheral iridectomy functions by deepening the anterior chamber owing to short-circuiting of the aqueous past the resistance of the iris. The operation is successful in narrow-angle glaucoma of the acute type, the tension being controlled without miotics in nearly all cases if the eyes are properly selected. When it is successful the eyes are completely normal, the disease being cured.

Intravenous Infusion of Typhoid Vaccine for Eye Diseases.

R. BUXEDA (*Arch. Ophth.*, September, 1952) describes the technique of continuous typhoid infusion. The infusion is usually started in the early morning with the patient fasting. Two mils of triple typhoid vaccine containing 1,000,000,000 typhoid organisms per mil are introduced into a flask containing one litre of sterile isotonic saline. This is run into the antecubital vein at a rate of 20 to 25 drops per minute. The infusion usually takes seven to ten hours. The treatment may be repeated within forty-eight to seventy-two hours. The author describes his results in 10 cases of irido-cyclitis, iritis, choroiditis, disciform keratitis and vitreous hæmorrhage. In all cases the reaction was fairly uniform, temperatures fluctuating between 99° and 101° F. The objective in mind is the production of a steady and uniform effect of killed typhoid organisms on the defensive mechanism of the body, along with the production of a sustained stimulation of the adrenal cortex which leads to the production of cortisone.

Association of Uveal Nævi with Skin Nævi.

A. B. REESE (*Arch. Ophth.*, September, 1952) describes four cases of nævi of the iris associated with ipsilateral skin nævi. He expresses the opinion that when a pigmented lesion of the iris is associated with excessively numerous or large ipsilateral skin nævi, it is likely to be a nævus. Although the majority of such lesions will not become malignant, there is always the possibility that the iris nævus will take on active growth and it is more likely to become malignant than the skin nævus. For this reason it may be advisable to excise all nævi of the iris. Complete local excision is usually possible.

Delayed Re-Formation of Anterior Chamber After Trephine Operations.

R. NACCACHE (*Brit. J. Ophth.*, August, 1952) describes a simple and effective method for dealing with the worrying complication of delayed formation of the anterior chamber after trephine operation. A silk thread is placed horizontally a few millimetres above the trephine hole and over the conjunctiva. The thread is anchored at both ends to the sclera in the fashion of a double mattress suture. The two anchoring points are placed beyond the conjunctival flap. The anterior chamber re-forms forty-eight hours after placing of the suture, which, however, is left in position for a further two or three days. The author recommends this procedure if the anterior chamber has not re-formed in ten days.

Lamellar Resection of the Sclera for Retinal Detachment.

MILTON L. BERLINER (*Arch. Ophth.*, November, 1952) writes a preliminary report on lamellar scleral resection. He states that the operation is indicated in cases in which shrinking and retraction of the retinal vitreous substance occur, especially when the retina has become fragile. The principle of scleral shortening lies in the attempt to adapt the sclera and the choroid to a displaced shrunken and shortened retina. In the author's opinion lamellar resection with diathermy coagulation should be performed as the primary procedure in these so-called bad cases. In myopic detachments with a tear in which improvement occurs with bed rest, the classical diathermy operation should be carried out. In aphakic detachment lamellar resection should be the primary procedure, even though one or more tears are found. The author outlines his technique and after-treatment.

Ophthalmic Manifestations of Temporal Arteritis.

GERALD PARSONS-SMITH (*Brit. J. Ophth.*, November, 1952) describes the various ocular manifestations of temporal arteritis and reports 15 cases. He states that temporal arteritis is a widespread arterial disease in which arterial

and local signs occur. The clinical features are anorexia, loss of weight, joint and muscle pain, pyrexia, painful arterial thrombosis and very severe headaches in elderly patients. Various ocular manifestations occur in about half the cases and can be divided into three groups: occlusion of the retinal artery within the optic nerve, ischæmic retrobulbar neuritis due to involvement of the retinal artery before it enters the optic nerve, and retinal phlebitis, photophobia and subretinal hæmorrhage. Blindness has been reported in both eyes.

Egg Membrane for Chemical Injuries of the Eye.

MAURICE CROLL AND LEO CROLL (*Am. J. Ophth.*, November, 1952) describe a method of treatment of chemical burns of the eye with egg membrane. They are of the opinion that from a clinical standpoint it would seem that the chemically injured conjunctiva is the main factor in causing scarring, ulceration and vascularization of the cornea. Insertion of egg membrane between the conjunctiva and cornea serves as a protective cover for the cornea. Acid burns of the eye are not so severe as alkali burns, since they are self-limiting, non-progressive and unpenetrating; and damage to the eyes is an initial damage that can, with few exceptions, be estimated fairly accurately when first seen. As a rule only the epithelium or superficial layers of the stroma are involved. Further penetration of acid is prevented by precipitation of corneal proteins. The aqueous is not affected, and secondary changes in the globe do not occur. Alkali burns are difficult to evaluate and are treacherous in their mode of action and progress; alkalis cause burns that are slowly progressive and are not self-limiting. They are destructive and penetrate into the anterior chamber, so that the aqueous becomes altered to a destructive medium. The authors describe in detail their preliminary treatment and operative procedure in the treatment of alkali burns with egg membrane.

OTO-RHINO-LARYNGOLOGY.

Common Disorders of the Vestibular System.

M. R. DIX AND C. S. HALLPIKE (*Ann. Otol., Rhin. & Laryng.*, December, 1952) present certain new information upon the subject of Ménière's disease and describe two other varieties of organic vertigo: one a disorder of the vestibular neurons; another a disorder of the otolith system in the labyrinth. They state that there is little to add on symptomatology to Ménière's original description of the disease which bears his name. More is to be made of distortion of hearing and of the exacerbations of tinnitus and deafness during the attacks. The caloric test results are abnormal in 94% of cases, and of these 20% show a directional preponderance towards the sound ear. In 54% there is a loss of canal sensitivity in the affected ear. In the field of cochlear tests, the phenomenon of loudness recruitment was invariably present in Ménière's disease. Speech audiometry tests reveal a loss of intelligibility out of proportion to the pure tone audiometric threshold loss. Since recruitment is characteristically absent in cases of eighth nerve tumour, it is concluded that the phenomenon is attributable to hair-cell disease. Microscopic material from a case of Ménière's disease shows very striking changes in the cells of Corti's organ of the affected side, while the cochlear nerve fibres and cells of the spiral ganglion are quite normal. In another group of cases the chief symptom is vertigo, usually paroxysmal, but cochlear signs and symptoms are absent. Here the symptoms seemed attributable to some form of organic disease confined to the vestibular apparatus. This condition has been termed "vestibular neuronitis". Over 100 cases have been studied at the National Hospital, Queen Square, London. The disorder chiefly affects the age group thirty to fifty years, without preference for sex. Apart from the absence of cochlear signs and symptoms it is distinguishable by the character of the vertigo, which consists of sudden and transient seizures accompanied by a sensation of blackout. In other instances no severe paroxysms may occur, and the disequilibrium may take the form of a feeling of "off balance" particularly during walking or standing or associated with head movements. In a fairly high proportion of cases the onset of the symptoms is associated with some kind of febrile illness or with evidence of infection of the ears, nose and throat. Otoscopic findings and results of cochlear function tests are normal. The caloric responses are consistently reduced, often grossly so, and often on both sides. In 47 cases caloric responses were abnormal on both sides; in 53 the abnormality was unilateral. Since the cochlea is spared it is thought that the lesion is central to the labyrinth. The condition is benign. The affected person responds well to treatment of focal sepsis when this is evident and generally recovers in the course of a few years. In a few cases reestablishment of the caloric responses has been observed. Clear evidence of antral or tonsillar infection was present in 24% of a group of 50 cases, and in 46% of the same group there was either a history of infective illness at the time of onset of the vertigo or significant evidence of an infective focus in the nose and throat. In all except three of a group of 16 cases regarded as vestibular neuronitis a significant reduction of the responses to galvanic stimulation was observed. This finding is considered as supporting the contention that the lesion is of Scarpa's ganglion or of the vestibular neurones central thereto. A third variety of organic vertigo is distinguishable on clinical grounds from Ménière's disease and from vestibular neuronitis. This type, first mentioned by Baranay in 1921 in a case of otolith disease, is characterized by paroxysmal vertigo and nystagmus and runs an essentially benign course. The hearing and caloric test results may be normal. Typically the story is that giddiness comes on when the patient lies down in bed. It can be demonstrated that certain changes in posture will precipitate the transient attacks and that return to the former posture may induce a further, but less severe, reaction. Some nausea and occasional vomiting may be experienced. Nystagmus of a rotatory type is usually found to occur with experimentally induced attacks and is directed to the undermost ear, but it is generally reversed if the patient is returned to the former upright position. Results of tests of cochlear function and of vestibular reactions have been found to be normal. In more than half of a series of 100 cases, there was substantial evidence of middle ear disease; in one-third such evidence was entirely absent. No evidence has been found of a neurological lesion. In some cases infective lesions such as of antra or teeth have been found, but in many cases these are absent. The evidence suggests that ear disease plays a part in the causation of the condition. The side of the ear disease does appear to be related to the direction of the nystagmus in a systematic manner. The lesion, if it is in one or other labyrinth, causes no other demonstrable disturbance of its function. Opportunity has occurred to examine the labyrinths in one such case. On one side degenerative changes were noted, including disappearance of the otolith membrane from the macula of the utricle. There were also infiltrative inflammatory changes in the connective tissue underlying the epithelium. Similar changes of lesser degree were noted in the macula of the saccule.

"Sulfamylon" in the Treatment of External Otitis.

K. L. DIEHL (*Arch. Otolaryng.*, January, 1952) states that "Sulfamylon" is a very effective bactericidal and bacteriostatic agent. It is a sulphanilamide derivative of somewhat altered structural formula. It is non-toxic and is active in the presence of para-aminobenzoic acid, blood, tissue juices and pus. The patient's ear is first thoroughly cleaned out and painted with 95% strength alcohol, which is allowed to dry. The canal wall is then painted over with a solution of 5% "Sulfamylon", and a wick soaked in the solution is left in the canal for six hours. After this the patient instills a 1% solution as drops into the canal every three hours. Of a series of 30 patients, 94% had definite improvement or cure in less than one week.

Palpation of Stapes for Fixation.

SAMUEL ROSEN (*Arch. Otolaryng.*, December, 1952) states that one cannot determine with absolute certainty if the stapes is completely fixed by any method except palpation of it with a probe, as may be done during the fenestration operation. Some inferential knowledge may be gained from pure-tone and speech audiometry. Under suitable premedication and local anæsthesia an incision is made around the lower half of the external auditory canal about six to seven millimetres external to the drum. The skin is separated from the canal as far as the drum, and thence the latter is lifted out of its sulcus and reflected upwards upon itself like an apron. The incudo-stapedial joint is thus readily exposed to view, so that the various parts may be tested for mobility with a probe. Afterwards the drum and skin of the canal wall are returned to their normal position. The patients are able to leave hospital the next day. The author suggests that conduction deafness due to adhesions may be dealt with in this manner, and it may be possible to render a partially fixed stapes foot plate more mobile, as seemed to occur in one of the author's five cases, and thus to restore hearing in a number of cases of otosclerosis.

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

LXXII.
CHILBLAINS.

JUST as it is observed in the study of the names of certain other human diseases, for example, "pellagra" derived from the Italian *pelle agra* meaning rough skin, so the word "chilblain" in its derivation reveals a suggestion of the nature of this very minor malady, a malady that afflicts humans generally, but not exclusively, during the winter season in the colder climates of the various regions of eastern Australia. Chilblains are confined to humans, no animal—horse, bovine, sheep, dog or cat—being known to suffer in the experience of those from whom inquiry has been made. Such inquiry has been made of scientifically trained local officers of the Commonwealth Scientific and Industrial Research Organization as well as of men who have had a life-long practical experience on grazing and farming properties. There is in the Bible no reference to the word, for which there is some cause for surprise, nor in the translations of the writings of Hippocrates has any reference been made to chilblains or to any other condition that could suggest it. One accepted explanation for this is that, in the warm Mediterranean littoral countries, the malady was not in evidence.

It is of introductory interest to trace the origin of the word. W. W. Skeats's "Etymological Dictionary of the English Language" states that "chilblain" is a blain caused by cold, literally "chill-blain", that is, a cold-sore, a sore caused by cold. Blain is a pustule, from the Anglo-Saxon blégen—a boil, a pustule. The form blégen is formed (by the diminutival suffix en) from the stem blag—a variation of blaw—to blow. It means that which is blown up—a blister.

And that is, in clinical fact, what a chilblain is, with numerous interesting variations of degree. The great majority of sufferers do not seek a doctor's advice and treatment. They treat their "chillies", as they thus refer, almost affectionately, to their annual winter disabilities, either by one or several of the many tried and, in some cases, proved effective home and "bush" remedies or, in a progressively increasing number of cases, because of successful advertising in the Press, by the tablets, mixtures and ointments available to the public over the counters of pharmaceutical chemists.

That exposure to cold is the immediate cause of chilblains is a straightforward observation in practice. Chilblains afflict sufferers in winter and not in the warm seasons; they occur much more frequently in cold climates than in warm localities. More chilblain sufferers amongst school children and young adults are known in New England than, for example, on the Darling Downs, and certainly in Brisbane, which has a mild winter climate, I can recall that in hospital experience there was not one report of a chilblain sufferer. My own observation is that amongst school children and also amongst university and teachers' college students, girls and young women outnumber boys and young men as patients; further, that in the still older adult population, men are very infrequent sufferers. Systematic inquiry of nursing sisters, who have the care, in their various boarding schools and residential hostels, of children of school age and also students attending tertiary educational institutions, has revealed that, amongst two thousand children in the school age-group, nine to sixteen years, there was an incidence in the winter of 1952 of 15%; and that amongst five hundred students in the college age-group, 17 to 22 years, there was during the same period an incidence of 5%.

Armidale's height above sea-level is 3350 feet and its distance from the nearest point on the coast of New South Wales is 101 miles. The winter is a long one from April to September; the temperature in the early morning through June, July and August is regularly below freezing point, fourteen degrees of frost being not unusual. There is each winter a small number of light falls of snow in the municipality, falls which leave the surrounding mountains and hills capped for four and five days.

The following first-hand account of chilblain experience is included to serve as the sample case history of the group of patients who come into the care of doctors. The patient is a senior nurse just about to complete her four years' course of training at the Armidale and New England District Hospital. In passing it is here stated that, from amongst the seventy nurses trained and in training at that institution, two suffered in the winter of 1952 from chilblains to such a degree that they sought medical assistance. It is one of these who has also supplied for inclusion in this article an account of her chilblain vicissitudes. She spent her schooldays in Guyra, whose altitude above sea-level is a thousand feet higher than that of Armidale. Regularly each winter she had chilblains on her feet; at no time on the hands, ears or nose. Her brothers suffered also from chilblains, having them on the ears and hands. Apropos of this observation, she has stated that her interested inquiry of schoolmates, of girl friends in her home and other towns and of her sister-nurses has prompted her observation that girls do not have chilblains on the ears. (This generalization has not been absolutely supported by the results of study of the site of chilblains in the schoolgirls and women students of Armidale. At least two schoolgirls were found to state that they regularly had chilblains on the ears.) For the relief of her chilblains during her school years our nurse applied a number of home remedies with varying degrees of relief, the most successful being household methylated spirit, which was swabbed on with cotton wool, followed by the application of an ointment which contained menthol chiefly. The chilblains did not break and did not disappear, though the symptoms were relieved, until the morning frosts ceased. Over the whole ten-year school period she on no occasion had "broken" chilblains.

During the first winter of her Armidale and New England District Hospital training her chilblains became much more severe to the point where they were disabling and compelled her to report "sick". Working in a centrally heated hospital, she observed that moving from warmer indoor through colder outdoor temperatures in order to reach the adjacent nurses' home was soon followed by a most uncomfortable aggravation of the chilblains. The backs of the legs became affected in addition to the toes, soles and heels. The nodules, now intolerably itching and painful, coalesced and involved the feet so widely that the only footwear possible was old, soft, amply fitting shoes. The pain was very great and the suffering very severe. At the backs of the heels and under the laces of the shoes the chilblains burst. She was compelled by her suffering and resulting inability to do her work to report "sick". She was removed from duty and treated. As the chilblains were now complicated by localized inflammation, she was given lounge rest, with the lower limbs elevated, penicillin tulle applied to the chilblains and three injections of "Calciostelin". At the end of two weeks the broken chilblains had healed so that she was fit to return to duty. During the remainder of that winter she suffered no chilblain discomfort greater than that from a few mild lesions.

During the following summer this nurse had *erythema nodosum* for which she received, for two weeks daily, injections of "Calciostelin" and for the following fourteen weeks bi-weekly injections of the same. In the next two winters she had no chilblains, though she worked in the same environment as before, a centrally heated hospital and a low out-of-doors temperature ranging down to fourteen degrees of frost. She also paid her regular home visits during these winter months to her one thousand feet higher town of Guyra where the frosts were more severe, the winds more biting and the snowfalls more heavy and more frequent. Her conclusion now is that the treatment she received for the *erythema nodosum* has proved to be a good prophylaxis for her chilblains.

This first-hand account from an experienced and competent nurse paints the overall clinical picture of chilblain manifestation. In a susceptible person during the winter months, the first evidence is a tingling, soon becoming an itching which is merely pleasantly uncomfortable or, more accurately, uncomfortably agreeable. The site of this is any one or more of toes, feet, backs of legs, fingers, hands, ears or nose. This itching is, in the next stage, succeeded by a more intense degree of discomfort which distracts the sufferer's attention from his or her normal activity and compels some attempt to gain relief. Usually this is most easily secured by immersing the affected part in water at the temperature of the body or a little higher. The chilblain is usually felt by the patient on coming to the warmth of an open fire, stove, electrical radiator, steam pipe or centrally heated building following exposure to the cold of an outdoor winter temperature. It also follows the wearing of wet clothing or footwear in games or other outside normal activities. The next stage is the definite interference with the patient's work by the continuing and increasingly intolerable itching. As a result of the scratching of the chilblain in an effort to get even momentary relief, the skin

surface is broken and there is now added the pain resulting from exposure of nerve endings. Following the coalescence of several chilblains in this stage competent medical attention is required. The few patients who have suffered in past winters from chilblains to this degree have impressed me by their reports of the intensity of their suffering. The majority of the 5% adult sufferers give a very light-hearted description of their annual winter disabilities. From May onwards they casually mention to their friends that "my chillies are beginning to show up" in much the same off-hand way as they would, if they were gardeners, as some are, announce in early spring that the weeds were showing through in their garden beds. They appear to accept their appearance in the same philosophical resigned manner as they regard an occasionally recurring pimple on the face—a mild nuisance that soon will disappear. Not so the genuine sufferer. A matron in charge of the health of New England University College students has stated that she still has no relief from her intense chilblain suffering in the Armidale winter. Her doctor's treatment has to date given her only partial relief. Her conclusion at the moment is that removal from the cause is her only hope of coping with her disability.

The experience of trained nurses in Armidale in charge of children at boarding schools and students at the university and teachers' colleges is as follows for the 1952 winter. At one boarding school for girls, from amongst the 285 boarders varying in age from ten to seventeen years, the number who reported chilblains was 43, approximately 15%. The chilblains were chiefly on the hands, some were on the feet and, in two girls, on the ears. Only one girl required to be referred to her doctor for treatment.

The sister at another boarding school treated the girls in her care successfully with nicotinic acid and vitamin *K* analogue tablets.

The survey of the older age groups elicited somewhat similar facts. During a chilblain inquiry of two tertiary educational residential institutions where trained nurses were always available to students, the following information was given. Of 400 students the number reporting their chilblains was six, or 1·5%. One of these six was a young woman from Bangkok; at no time, even in summer time, did she feel comfortably warm and in winter she was "paralysed" by the cold. No student had chilblains on the ears.

The conclusions arrived at after a survey of (*a*) two thousand children of school age attending all types of schools, (*b*) four hundred university and teachers' college students, the majority of whom live in residential hostels, (*c*) seventy hospital nurses, trained and trainees, and (*d*) a small number of adults of a rural community with a central population of 8000 and an additional district population of 10,000 are as follows:

1. A very small percentage of these groups suffer from chilblains.

2. Only a handful of these sufferers need a doctor's treatment.

3. Home and over-the-counter remedies are efficacious in almost 100% of cases.

4. The challenge to the medical profession—its practitioners and its researchers—is to find the satisfactory answer to the problem of prevention of chilblains in the known susceptibles.

Because calcium has been proved to be a reliable prophylactic and, in the great majority of patients, a satisfactory cure, our attention is directed mainly to its use in therapy; but there is at least one other method of treatment referred to later in this article which is worthy of attention.

It is stated that the therapeutic action of the calcium ion is to reduce cell permeability and fluid transudation and that the frequent immersion of the hands, feet or ears in hot water, together with the injection of calcium daily, will generally effect a disappearance of chilblains. Why cell permeability and fluid transudation should need reduction in fifteen out of every hundred schoolgirls at an Armidale boarding school and in two of every hundred university and teachers' college students is a challenging question, the answer to which does not present itself in clinical practice. Laboratory workers may, sooner or later, direct practitioners along the path that leads to this answer; careful observers and logical thinkers amongst general and special practitioners of medicine may from their ranks disclose another Mackenzie, or another Gregg, who will be the pathfinder to direct us all to the answer.

Approaching the conclusion of this article in a somewhat more orthodox style, I would state that there is a special predisposition to chilblains about which nothing is known for certain. Age is a factor, and environment is important. The treatment is varied.

First of all, reference must be made to what may be described as home or bush remedies. Only some of these need be mentioned. Some sufferers take a handful of frost or snow and rub the affected parts vigorously with it. Others place the feet or hands in warm water, thinking the remedy is more satisfactory when the water has been strained from boiled potatoes. Some sufferers rub the affected region with eucalyptus oil. Others soak the feet or hands in hot water in which washing soda has been dissolved, and others again rub the chilblain with tincture of iodine or methylated spirits. Some sufferers go so far as to scarify the bursting chilblain with a safety razor blade, and claim that relief is gained from the release of enclosed fluids and the resulting lowering of tension. We find that this is not an uncommon practice. These more or less desperate remedies have been grasped at by sufferers who have had only their own resourcefulness to prompt, and their own resources to aid, them. I think that warmth and the administration of calcium give most relief.

In recent years, calcium and calcium tablets and mixtures have been used in varying forms, and many persons buy these remedies directly from the pharmacist. In recent years, too, vitamin *K* analogue and nicotinic acid tablets have been used more or less extensively with an increasing belief in their efficiency. A preparation known as "Pernivit" sold in tablet form has won increasing favour.

The treatment generally given by a doctor consists simply of the use of calcium by injection, "Calciostelin" being the preparation generally used. In the survey of 3000 cases of chilblains, it was noted that one boy was reported as having undergone ramisection in order to obtain relief. Only one patient in the series was reported as having failed to receive relief from treatment by a medical practitioner. In this instance, the "cure" or "relief" came to an ex-army sister only with the departure of winter and the advent of spring—a long-awaited event.

R. J. JACKSON,
Armidale, New South Wales.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held at the Royal North Shore Hospital of Sydney, Crow's Nest, New South Wales, on April 23, 1953. The meeting took the form of a series of clinical demonstrations by members of the medical and surgical staffs of the hospital.

Progressive Myositis Fibrosa.

DR. I. A. BRODZIAK showed a man, aged thirty-eight years, suffering from progressive *myositis fibrosa*. In 1923, at the age of eight years, the patient had noticed the gradual onset of an equinus deformity of both feet, causing him to walk on his toes. One year later, it was corrected surgically, but the original deformity recurred. At the age of twelve years the patient noticed that his legs were weak, especially when he walked upstairs, and four years later he noticed that his legs were wasting, and found that he had to put his hands on his knees to rise from the sitting posture. At the age of eighteen years he found that he could not extend his elbows fully. That was followed by flexion deformities of the fingers and limitation of abduction of the shoulder joint. About the same time he found that he could not flex his neck or back or fully open his mouth. Those symptoms persisted for ten years with progressive limitation of movement of the knees, ankles, elbows, shoulders, spinal column and small joints of the hands, together with wasting of all muscle groups. At no time had there been any pain, tenderness or swelling of any muscle groups, nor had there been any skin lesions except varicose ulcers. Other illnesses from which the patient had suffered included concussion, measles, malaria and gonorrhœa. There was no family history of muscular or nervous disease. The patient was thin and walked with crutches. His feet had a *pes equinus* deformity. All muscle groups of the extremities were wasted and firm, and there was no evidence of recent loss of weight. Dr. Brodziak detailed the range of movements of the patient's individual joints, as well as the respiratory excursion dependent on the intercostal muscles. He pointed out that there was no tenderness of muscles, nor were any nodules or

lumps palpable in the muscle groups. The joints were not tender, and movement was painless. No fibrillation was present, nor were there any abnormal nervous signs except absent or diminished reflexes. The muscles of expression were not involved.

Bronchial Carcinoma Involving the Liver.

Dr. Brodziak, with DR. L. S. LOEWENTHAL, then presented details of a case in which a tumour thought during life to be in the liver was found at autopsy to be in the lung. The patient, a man, aged forty-six years, had fatigue, loss of energy, anorexia and slight loss of weight in December, 1952, and then developed right-sided pleurisy, with exertional dyspnœa. He had no significant previous symptoms apart from hæmatemesis two years before. Born in South Africa, he had lived in Sydney for twenty-seven years apart from time spent in the Philippines during World War II. When admitted to hospital on December 31, 1952, the patient had a large right-sided pleural effusion and a temperature of 99° F. Other systems were apparently normal. The liver was not palpable. Blood examination revealed a hæmoglobin value of 10·6 grammes *per centum* and a moderate degree of leucocytosis. The Mantoux reaction was positive, but material obtained by gastric lavage contained no acid-fast bacilli. The result of the Casoni test was negative. One pint of blood-stained effusion was aspirated from the chest; the supernatant fluid was xanthochromic and contained 110 milligrammes *per centum* of sugar, and five grammes *per centum* of protein, but no acid-fast bacilli or cancer cells. Evidence of an effusion persisted, and the patient developed pyrexia associated with further leucocytosis. X-ray examination showed evidence of an effusion, and there appeared to be a rounded area of increased density at the right lung base anteriorly. Further attempts at aspiration were unsuccessful, and penicillin therapy did not reduce the fever. No other source of infection could be found. A diagnostic pneumoperitoneum was induced, and the rounded mass previously thought to be in the lung was considered to be below the diaphragm. Needling of the liver produced no pus. On February 24, 1953, laparotomy was performed by Dr. Loewenthal. The liver was enlarged, and adhesions were present between the liver and diaphragm. The liver was again needled, but no abscess cavity was discovered. Except for slight splenic enlargement, no other abnormality could be found. Liver biopsy revealed mitotic activity suggestive of the presence of a malignant hepatoma. After operation, the patient remained pyrexial and his condition steadily deteriorated. The liver was greatly increased in size. The axillary and supraclavicular lymph glands enlarged, and on March 16 biopsy of a gland in the right axilla showed it to be largely replaced by a malignant tumour which histologically resembled the section of liver obtained at biopsy. At autopsy the tumour, previously considered to be in the liver, was found to be in the lung; it was a bronchial carcinoma. The pleura was covered with malignant tissue, which had also involved the liver, peritoneum and mesenteric and mediastinal glands.

Meningitis of Uncertain Ætiology.

DR. F. A. E. LAWES showed a man, aged twenty-nine years, who for the past fourteen months had been employed on a pig farm. During his period of employment mortality amongst the pigs had been high, but the cause of death was not known. The patient had been admitted to hospital on March 14, 1953, with a history of malaise, easy fatigability, loss of appetite and loss of weight for three months. Previously he had enjoyed robust health. On March 4, 1953, he had developed a dull, aching pain in the right side of the chest; it was intermittent and lasted for two days. On March 8 he developed a severe generalized headache, worse in the frontal area, weakness in all his limbs, generalized aches and pains, and severe dull pain in the lower part of his back and buttocks. His bowels did not act between March 10 and 14. At the time of admission to hospital, the patient had a raised temperature and was dehydrated. He had severe neck rigidity, but Kernig's sign was absent. All tendon reflexes were present and symmetrical, but the knee jerks were diminished. The capillary and abdominal reflexes and the plantar responses were normal. The margins of both optic disks were blurred. No abnormalities were found elsewhere in the patient's nervous system or in other systems. Lumbar puncture produced an opalescent fluid under increased pressure. The Queckenstedt test produced a normal response. The fluid contained three erythrocytes, 234 polymorphonuclear cells and 66 lymphocytes per cubic millimetre. No organisms were grown on attempted culture for forty-eight hours, and no acid-fast bacilli were detected by smear examination or attempted culture. The fluid contained 45 milligrammes *per centum* of protein, 710 milligrammes *per centum* of chloride, and 63 milligrammes *per centum* of glucose. The patient was treated with bed rest and appropriate sedation and analgesics. No specific therapy was given. Serum agglutination tests produced a "++" response in a serum dilution of 1:300 for *Leptospira icterohæmorrhagiæ* and *L. pomona*, in a dilution of 1:100 for *L. canicola*, and in a dilution of 1:30 for *L. australis A*. The response was negative from a dilution of 1:10 for *L. australis B* and *L. mitis*. Other investigations failed to produce a significant response.

Symptomatic Acquired Hæmolytic Anæmia.

Dr. Lawes also showed a woman, aged sixty-four years, who had been admitted to hospital in January, 1953, with symptoms and signs indicative of an acute respiratory infection. She was dehydrated and wasted, with shallow varicose ulcers on the left leg and decubitus ulcers over the sacrum. Signs of consolidation were present at the right lung base. The patient was given a diet of high protein content, with vitamin supplements and free fluids, and penicillin. Her symptoms and raised temperature quickly subsided, but her chest signs remained. One week later, her temperature again rose. Exploratory needling of the chest in the area of greatest dulness produced neither fluid nor pus, and the temperature returned to normal with aureomycin therapy. Over the next ten weeks the pyrexia recurred several times, the chest signs and X-ray appearances changed very little, and further exploratory needling failed to produce pus. On March 28, 1953, significant improvement began in relation to the chest signs and X-ray appearances. Attempts at isolating pathogens from the sputum or gastric washings, and the Mantoux and Casoni tests all produced negative results. Referring to the hæmatological features, Dr. Lawes said that in the early acute stages of the illness, leucocytosis had occurred with a cell count of 28,750 per cubic millimetre, and a relative lymphopenia. Within a fortnight the count had returned to normal, and had remained so apart from transient leucocytosis during one of the attacks of pyrexia. Immature leucocytes had been present when most of the counts were made. The blood platelets throughout had appeared to be normal, in both number and appearance. The hæmoglobin value had throughout remained around nine grammes *per centum*. An abnormal degree of variation in size of the erythrocytes had been constantly present with a tendency towards spherocytosis. Polychromasia had been present in an occasional cell. Reticulocytes had varied in number from nil to 3%. A red cell fragility test revealed that hæmolysis commenced at 0·47% sodium chloride solution, and was complete at 0·3% sodium chloride solution. An indirect Coombs test, in which the patient's serum was used against test cells, yielded negative results. The red cells of the patient appeared to be a mixture of Rh-positive and Rh-negative cells. On February 23, icterus of the scleræ was noticed for the first time. The serum bilirubin content was then 3·2 milligrammes *per centum*, bile pigments were absent from the urine, the urinary urobilinogen content was greatly increased, the thymol turbidity test result was 2·5 units, the serum alkaline phosphatase content was 35·7 units, and the serum protein values were normal. The clinical appearance of jaundice rapidly cleared, but the serum bilirubin content had remained slightly raised until the past week, when it returned to normal. At no time were bile pigments detected in the urine. The serum alkaline phosphatase level remained moderately raised as long as the serum bilirubin content was raised, but it had now returned to normal. The serum protein values and thymol turbidity had remained normal throughout.

Chronic Hæmolytic Anæmia.

DR. DOUGLAS ANDERSON showed a single woman, aged thirty-six years, who had suffered from chronic hæmolytic anæmia from childhood, and had recently been made well by means of splenectomy. No other member of her family had had the disease so far as could be discovered, and no hæmolytic crisis had occurred during the twenty-three years the anæmia was known to have been present, but during the whole of that time she had had only about half the normal number of red corpuscles. Dr. Anderson said that classification of chronic hæmolytic anæmia into "familial" and "acquired" types was illusory as a means of clarifying therapeutic indications, though a family history of the disease could be taken as a good indication for splenectomy, as were long chronicity of the disease, onset in early life, the presence of spherocytes and excessive osmotic fragility of the red corpuscles. The indications were weakened by a positive Coombs test result. Dr. Anderson said that in the present case, positive reactions had been obtained in the blood to the Wassermann and Eagle tests. These he took to be "false positives", probably due to the presence of

abnormal globulin in the patient's plasma. He considered that cortisone had no place in the treatment of hæmolytic anæmia, except in endogenous types in which the disease was exacerbated and intravascular hæmolysis could be demonstrated. Much fundamental knowledge about chronic hæmolytic anæmia was still lacking. The cause of spherocytosis was unknown, as was the reason for the osmotic fragility of the red corpuscles, and the two phenomena were not correlated. Other obscure features were the part played by the spleen in the pathogenesis of the disease, its mode of selecting red corpuscles for destruction, its part in the production of hæmolysins, the nature of the so-called hæmolytic crises and, in particular, the reason why reticulocytes often became less numerous in the circulating blood and spherocytes more numerous in such episodes. The chemistry and physical chemistry of the hæmoglobin and of the plasma proteins did not appear to have been studied in relation to the disease.

Obscure Abdominal Pain.

DR. H. HUNTER JAMIESON showed a man, aged sixty-three years, who had been admitted to hospital in September, 1952, with a history of gradual increase in the size of his abdomen for six months. For four days he had been passing mucus *per rectum*. For two days he had had pain in the left side of the abdomen, severe and fairly continuous, and burning in quality; it waxed and waned, but just prior to his admission to hospital it had become very severe and radiated down the left groin and testicle. He had vomited once on the day of admission to hospital. His bowels were acting, though only a small amount of fæces was passed. Examination of the patient revealed that his abdomen was obese, swollen and bulging in the lumbar regions, with considerable shifting dulness. No tenderness or rigidity or palpable masses were detected. Plain X-ray examination of the abdomen revealed considerable distension of the large intestine around the hepatic flexure, but no fluid levels were seen to indicate obstruction. Enemata produced only a fluid result. *Streptococcus viridans* was grown from the urine, which contained erythrocytes and occasional pus cells. Laparotomy revealed only diverticulosis and an uncomplicated Meckel's diverticulum of the wide-based type, with increase in omental fat. Convalescence was complicated by paralytic ileus which responded only slowly to treatment, and was followed by profuse diarrhœa, for which no cause could be found. However, the condition gradually subsided.

Argentaffin Carcinoma of the Small Bowel.

Dr. Jamieson then showed a man, aged sixty-two years, with a nine months' history of anorexia and loss of weight. For three months the patient had had colicky abdominal pain, situated chiefly in the left iliac fossa and below the umbilicus, made worse by eating and unrelieved by anything. He suffered from belching and audible borborygmi. For one week he had had alternating constipation and diarrhœa, with aggravation of the abdominal pain. For two days he had been vomiting brownish fluid. On examination, the patient's abdomen was found to be distended and tender, with an increase in bowel sounds. A plain X-ray examination showed distended loops of bowel with fluid levels. At laparotomy, about two feet of small intestine, dilated and partly gangrenous, were resected. The bowel appeared to be bound down to a matted mass of retroperitoneal lymph glands, and was partly twisted. Pathological examination of the resected specimen revealed it to be an argentaffin carcinoma which had spread to the lymph nodes. Convalescence was stormy and complicated by paralytic ileus, a deep venous thrombosis, lateral popliteal nerve palsy, a sacral bed sore, and *alopecia areata*.

Idiopathic Megacolon.

Dr. Jamieson's last patient, a man, aged sixty-eight years, had been admitted to hospital in August, 1952, with a two years' history of precipitancy of defæcation and micturition. He had then a soft, distended abdomen, but rectal examination revealed no abnormality. Plain X-ray examination of the abdomen showed a colon distended with gas, but no fluid levels and no obvious obstruction; it was considered that if obstruction was present, it should be in the sigmoid colon. No significant further information was produced by barium enema examination, full blood count, excretion pyelography and sigmoidoscopy. At laparotomy the left half of the transverse colon and the upper half of the descending colon were found to be grossly dilated and redundant. The splenic flexure and descending colon were not fixed. One week later resection of the colon was performed with end-to-end anastomosis. The patient made a slow but complete recovery. The pathologist reported that the specimen of colon removed was considerably dilated throughout its whole length, the circumference towards the centre measuring 23 centimetres. In sections taken from each end of the length of colon the ganglion cells of both Auerbach's plexus and Meissner's plexus were microscopically visible and appeared normal. The only abnormality seen was mild fibrosis of the submucous layer.

(To be continued.)

WESTERN AUSTRALIAN BRANCH NEWS.

Post-Graduate Programme.

THE following is the post-graduate programme of the Western Australian Branch of the British Medical Association for the remainder of the year:

Wednesday, August 19, 1953: Clinical evening presented by general practitioners at the Royal Perth Hospital at 8.30 p.m.

September 7 to 12, 1953: Pædiatric week, during which the profession will be given the opportunity of hearing Professor Lorimer Dods and Dr. T. Y. Nelson, both of Sydney, and Dr. Ronald McKeith, a London pædiatrician.

Wednesday, October 21, 1953: Clinical evening at the Fremantle Public Hospital.

Wednesday, November 25, 1953: Talk by the City Coroner, Mr. R. P. Rodriguez, on "The Coroner and the Medical Profession" at the Royal Perth Hospital at 8.30 p.m.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

SMALLPOX IN VICTORIA.[1]

[*The Australian Medical Gazette*, Melbourne, February 15, 1869.]

WHENEVER the history of the present outbreak of smallpox in Victoria shall come to be written, although heaven knows the subject is serious enough, the story will not be altogether devoid of that ludicrous element so often found intermingled with the gravest concerns of human life. We have no doubt it will be a matter of surprise and wonder to our medical brethren in other parts of the world, how it could come to pass that so distinctive a disease and one characterized by such a peculiar set of symptoms, as smallpox when full developed, could not immediately be recognized by any practitioner of average education and capacity seeing that Sir Thomas Watson in his classical work on the practice of medicine, volume 2, page 856, speaking of variola, says, "when smallpox is full formed it cannot be mistaken for any other complaint". Now many of the sufferers during the present complaint had the complaint so well developed that they may be described as typical cases of smallpox, no symptom necessary to a complete picture of the disease, as drawn by the best writers on medicine, being absent.

Had ordinary care and intelligence been exercised on the first arrival here of the "Avondale" the existence of smallpox on board should and ought to have been discovered: and even had the chief medical officer at a later period attended to the repeated warnings given to him in all probability the scourge would have been stamped out and Victoria would not now have the fell disase apparently acclimatized within her borders and would not now be reaping the bitter fruits arising from the chief medical officer's wilful and dogmatic rejection of advice. It must be satisfactory to the public as well as creditable to the capacity of the profession in Victoria that the practitioners of the city and suburbs were from the first—with the exception of a very few, who, following the fashion of the hour, were inclined to pin their faith on the newly arrived lights "of some eminence"—nearly unanimous in approving the disease to be smallpox. Perhaps it may be well, before concluding, to take a rapid glance at some of the opinions—although some of them were "more

[1] From the original in the Mitchell Library, Sydney.

amusing than instructive"—advanced on the nature of the disease during the present epidemic. At first the public and the profession were gravely and persistently informed that the disease was the mild and harmless chickenpox, this view being put forward principally by the chief medical officer and his supporters. This position being found untenable, we were next authoritatively told "ex cathedra" that there was no essential difference between variola and varicella, these being, in fact, mere modifications of the same disease. Then followed, in succession, a number of other opinions equally ridiculous, such as that the disorder was neither chickenpox nor smallpox but an entirely new complaint, differing from both. All these various hypotheses, although wholly worthless, were nevertheless found very serviceable in masking the incapacity of their propounders, who, having for a while sneered and scoffed at men better informed than themselves, have a length been compelled with a very bad grace indeed to admit the disease to be genuine smallpox, whether modified or not makes little difference, as the modified form may give rise to the most malignant.

Correspondence.

THE TREATMENT OF CHRONIC RHEUMATISM WITH "BUTAZOLIDIN".

SIR: Regarding "Butazolidin", the following is of interest. A young female patient with chronic rheumatoid arthritis was placed on "Butazolidin", 200 milligramme tablets three times a day, and the patient showed an immediate improvement. White blood cells before starting treatment were 12,500 per cubic millimetre. After two weeks a further white blood cell count showed 2000 per cubic millimetre with patient quite well save for slight diarrhœa. The drug was discontinued, and one week later white blood cells had risen to 10,000 per cubic millimetre. The patient is now on 200 milligramme tablets once daily, and white cell count has remained around this latter figure. Differential white cell counts were within normal range for all the above readings. This only stresses again the fact that white blood cell counts should be carried out on all patients before and during "Butazolidin" treatment.

Yours, etc.,

William Street, Condobolin, New South Wales.
K. J. FITZGERALD, M.B., B.S.

July 12, 1953.

FOLIC ACID AND REPRODUCTIVE EFFICIENCY.

SIR: It has been shown that the anti-folic acid factor is detrimental to the fœtus *in utero*—therefore folic acid should be beneficial. The difficulty is to find a numerical factor of that deficiency apart from the presumptive history. If a single lymphocyte count is taken as this standard, how does one interpret the following serial counts from the same patient in my series of cases—actual counts or percentage on which date?

Date.	Total Leucocytes.	Lympho-cytes.	Per-centage.	Gestation.
December	8500	1785	21	24 weeks.
February 23 ..	9900	1683	17	32 weeks.
February 25 ..	7500	1350	18	
February 27 ..	6100	854	14	
March 2	8900	2848	32	
March 4	8500	1530	18	
March 6	7000	1470	21	33 weeks.

Other cases—to be published later—show a familiar variation on repeated counts during the pregnancy. My compliments to the authors[1] on their excellent end results.

Yours, etc.,

12 Collins Street, Melbourne, C.1.
W. J. RAWLINGS.

July 6, 1953.

[1] Dr. Rawlings is referring to the paper by D. F. Lawson, C. N. De Garis and J. H. Bolton in the issue of June 13, 1953.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Numbers 38 and 41, of June 18 and July 2, 1953.

NAVAL FORCES OF THE COMMONWEALTH.

Permanent Naval Forces of the Commonwealth (Sea-Going Forces).

To be Surgeon Captain.—Surgeon Commander James Martin Flattery, O.B.E.

Emergency List Promotion.—Surgeon Lieutenant Thomas Bowen Ready is promoted to the rank of Surgeon Lieutenant-Commander, dated 25th August, 1951.

Citizen Naval Forces of the Commonwealth.

Royal Australian Naval Reserve.

To be Surgeon Commander.—Surgeon Lieutenant-Commander Sidney Arnold Sewell.—(Ex. Min. No. 75—Approved 25th June, 1953.)

Royal Australian Naval Volunteer Reserve.

Promotions.—Surgeon Lieutenant (Surgeon Lieutenant-Commander (Acting)) Rex Vivian Blaubaum is promoted to the rank of Surgeon Lieutenant-Commander, dated 25th May, 1951. Surgeon Lieutenant Colin Alfred Cameron Galbraith is promoted to the rank of Surgeon Lieutenant-Commander, dated 16th November, 1952.

AUSTRALIAN MILITARY FORCES.

Australian Regular Army.

Royal Australian Army Medical Corps.

3/40112 Captain G. A. Rutherford is appointed from the Regular Army Special Reserve, and to be Captain, 19th January, 1953, with a Short Service Commission for a period of one year.

3/40113 Captain (provisionally) P. J. C. Stretton is appointed from the Active Citizen Military Forces, and to be Captain, 15th April, 1953, with a Short Service Commission for a period of one year.

2/40153 Captain (provisionally) R. H. Higham is appointed from the Active Citizen Military Forces, and to be Captain, 17th April, 1953, with a Short Service Commission for a period of one year.

Citizen Military Forces.

Northern Command: First Military District.

Royal Australian Army Medical Corps (Medical).—The provisional rank of 1/13388 Captain C. G. D. Clarke is confirmed.

Eastern Command: Second Military District.

Royal Australian Army Medical Corps (Medical).—2/221713 Major D. C. Williams is appointed from the Reserve of Officers, 25th March, 1953, and is borne supernumerary to the authorized establishments of Majors, with pay and allowances of Captain (at own request). The following officers are appointed from the Reserve of Officers: 2/206954 Major W. D. Sturrock, 29th April, 1953, and 2/61552 Honorary Captain R. H. Keatinge, and to be Captain (provisionally), 22nd April, 1953. The following officers relinquish the provisional rank of Captain and are transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (2nd Military District) in the honorary rank of Captain: 2/130104 K. H. S. Cooke, 23rd February, 1953, and 2/107771 R. J. Jennaway, 24th March, 1953. To be Captains (provisionally): 2/57599 John Frederick Parle, 30th March, 1953, 2/121993 John Beveridge, 12th May, 1953, and 2/101562 John Vincent Roche, 19th May, 1953.

Southern Command: Third Military District.

Royal Australian Army Medical Corps (Medical).—3/101827 Honorary Captain J. S. Crosbie is appointed from the Reserve of Officers, and to be Captain (provisionally), 5th May, 1953.

Central Command: Fourth Military District.

Royal Australian Army Medical Corps (Medical).—4/31952 Captain R. A. Kenihan ceases to be seconded for post-graduate studies in the United Kingdom, 16th March, 1953.

Western Command: Fifth Military District.

Royal Australian Army Medical Corps (Medical).—5/34602 Captain (provisionally) F. A. Murphy relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (5th Military District) in the honorary rank of Captain, 8th May, 1953.

Tasmania Command: Sixth Military District.

Royal Australian Army Medical Corps (Medical).—6/15247 Captain (provisionally) A. S. Wood relinquishes the provisional rank of Captain and is transferred to the Reserve of Officers (Royal Australian Army Medical Corps (Medical)) (6th Military District) in the honorary rank of Captain, 15th May, 1953. To be Colonel, 25th May, 1953: 6/15311 Lieutenant-Colonel (Temporary Colonel) P. Braithwaite, E.D.

Reserve Citizen Military Forces.

Royal Australian Army Medical Corps.

1st Military District: To be Honorary Captain, 22nd May, 1953.—Noel Stanley Glover.

2nd Military District: To be Honorary Captains.—Alan Paine Skyring, 13th April, 1953, James Marland Nield, 13th May, 1953; Gordon Thomson Archer, 19th May, 1953; and David John Hansman, 22nd May, 1953.

3rd Military District: To be Honorary Captains.—Peter Ebeling and Maxwell George Whiteside, 8th May, 1953, and William Scott Mitchell, 13th May, 1953.

1st Military District.—The following officers are retired, 1st July, 1953: Honorary Captains H. H. Power and F. F. Pincus.

2nd Military District.—Honorary Captain J. A. Thoms is retired, 1st July, 1953.

3rd Military District.—The following officers are retired, 1st July, 1953: Honorary Captains A. G. H. Springthorpe, J. M. Sleeman, M. C. Patrick, H. C. Worch, W. A. Baird and J. G. Cameron.

4th Military District.—The following officers are retired, 1st July, 1953: Honorary Captains K. M. Texler, R. J. DeN. Souter and E. McLaughlin.

The following officers are placed upon the Retired List within Military Districts as shown, with permission to retain their rank and wear the prescribed uniform, 1st July, 1953:

1st Military District.—Major (Honorary Lieutenant-Colonel) *H. W. Anderson, Major R. A. Maxwell,* Captains W. F. Batson, V. C. Byrne and A. E. Mason.

2nd Military District.—Lieutenant-Colonels (Honorary Colonels) D. B. Loudon and *W. H. Ward, E.D., Majors* C. J. B. Armstrong, C. F. A. De Monchaux, J. K. Harbison, R. St.J. Honner, L. W. Tunley and F. O. B. Wilkinson, Captains M. H. Elliot-Smith, W. Moppett and *A. F. Smith,* and Lieutenant S. H. Palmer.

3rd Military District.—Lieutenant-Colonel (Honorary Colonel) M. A. Rees, Lieutenant-Colonels K. C. Ross and T. U. Ley, Majors R. F. Phillips, G. G. B. Bolleau, R. C. E. Brodie, E. W. Casey, H. E. Pearce, E.D., F. J. Niall, Captain (Honorary Major) R. F. May, Captains G. E. Forman, R. McG. Laidlaw, D. C. Lear, R. Southby and G. J. McC. Stoney.

4th Military District.—Lieutenant-Colonel (Honorary Colonel) L. G. Male, E.D., Lieutenant-Colonel F. R. Hone and Captain C. W. Friebe.

6th Military District.—Captains L. A. Cramp and H. I. Gibb.

Royal Australian Air Force.

Permanent Air Force: Medical Branch.

Francis Edward Williams (039459) is appointed to a short service commission, on probation for a period of twelve months, 8th April, 1953, with the rank of Flight Lieutenant.

The resignation of Flight Lieutenant J. Q. McCubbin (036120) is accepted, 22nd May, 1953.

Squadron Leader (Acting Wing Commander) C. W. Fitton (033030) is promoted to the rank of Wing Commander.

No. 22 (City of Sydney) Squadron.

Kenneth Tweedale (0211512) is appointed to a commission, 7th February, 1953, with the rank of Flight-Lieutenant.—(Ex. Min. No. 58—Approved 4th June, 1953.)

Active Citizen Air Force: Medical Branch.

The following Flight Lieutenants are transferred to the Reserve: J. W. Walsh (051337), 27th January, 1953, T. D. Bourke (051338), 6th April, 1953.—(Ex. Min. No. 66—Approved 25th June, 1953.)

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED JUNE 20, 1953.[1]

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	..	6(5)	..	..	..	..	..	..	6
Amœbiasis	..	..	..	..	..	..	..	..	..
Ancylostomiasis	..	..	6	..	..	..	..	..	6
Anthrax	..	..	..	..	..	..	..	..	..
Bilharziasis	..	..	..	..	..	..	..	..	..
Brucellosis	..	1	..	..	..	..	..	..	1
Cholera	..	..	..	..	..	..	..	..	..
Chorea (St. Vitus)	..	..	..	..	..	..	..	..	..
Dengue	..	..	..	..	..	..	..	..	..
Diarrhœa (Infantile)	..	2	15(14)	..	..	..	..	..	17
Diphtheria	12(8)	2(1)	4	..	3(3)	2(1)	..	..	23
Dysentery (Bacillary)	..	..	1	1(1)	2(1)	..	..	..	4
Encephalitis	..	..	..	..	..	..	..	..	..
Filariasis	..	..	..	..	..	..	..	..	..
Homologous Serum Jaundice	..	..	..	..	..	..	..	..	..
Hydatid	..	1	..	..	..	..	..	..	1
Infective Hepatitis	..	14(5)	..	..	8(6)	..	..	..	22
Lead Poisoning	..	..	..	..	..	..	..	..	..
Leprosy	..	..	1	..	..	..	..	..	1
Leptospirosis	..	..	3	..	..	..	..	..	3
Malaria	..	..	..	..	2(2)	..	..	..	2
Meningococcal Infection	2(2)	4(2)	1(1)	..	1(1)	..	..	..	8
Ophthalmia	..	..	..	..	..	..	..	..	..
Ornithosis	..	..	..	..	..	..	..	..	..
Paratyphoid	..	..	..	..	..	..	..	..	..
Plague	..	..	..	..	..	..	..	..	..
Poliomyelitis	5(4)	4(0)	..	5(5)	..	..	..	..	14
Puerperal Fever	..	..	1(1)	..	..	..	..	..	1
Rubella	..	13(9)	..	..	8(3)	..	..	..	21
Salmonella Infection	..	..	..	..	1(1)	..	..	..	1
Scarlet Fever	12(11)	89(68)	4(2)	6(2)	1(1)	..	..	..	112
Smallpox	..	..	..	..	..	..	..	..	..
Tetanus	..	..	..	..	1(1)	..	..	..	1
Trachoma	..	..	..	..	..	..	..	..	..
Trichinosis	..	..	..	..	..	..	..	..	..
Tuberculosis	35(23)	12(6)	10(5)	..	8(6)	3(2)	..	..	68
Typhoid Fever	..	1(0)	..	..	..	..	..	..	1
Typhus (Flea-, Mite- and Tick-borne)	..	..	2	2	..	..	..	..	4
Typhus (Louse-borne)	..	..	..	..	..	..	..	..	..
Yellow Fever	..	..	..	..	..	..	..	..	..

[1] Figures in parentheses are those for the metropolitan area.

Air Force Reserve: Medical Branch.

The following are appointed to commissions with the rank of Flight Lieutenant: Richard Francis Orr Colahan (257897), 31st December, 1952, Martin Lindsay Waugh (268013), 28th January, 1953.

Flying Officer J. Scott-Findlay (424401) is transferred from the General Duties Branch, 26th January, 1953, with the rank of Flight Lieutenant.

The following former officers are appointed to commissions with rank as indicated: (Squadron Leader (Temporary Wing Commander)) D. McK. McNab (033032), 26th March, 1953; (Flight Lieutenant) M. M. McKeown (255912), 9th April, 1953.

Flight Lieutenant M. L. Waugh (268013) is granted the acting rank of Squadron Leader, 28th February, 1953.

Medical Prizes.

THE STAWELL PRIZE.

THE Medical Secretary of the Victorian Branch of the British Medical Association has announced that the Stawell Prize for 1952 has been awarded to Dr. Alfred J. Barnett and Dr. J. R. E. Fraser for their essay on peripheral vascular disease.

MEDICAL WOMEN'S SOCIETY PRIZE.

THE honorary secretary of the Medical Women's Society of New South Wales has advised that the Medical Women's Society Prize for 1952 was divided between Dr. Grace Cuthbert-Browne for her "Report of Studies and Observations Made during Tenure of a World Health Organization Fellowship, 1950-51" and Dr. Jean Palmer for her paper on "The Management of Hypertension with Hexamethonium Bromide".

Australian Medical Board Proceedings.

NEW SOUTH WALES.

THE following have been registered, pursuant to the provisions of the *Medical Practitioners Act*, 1938-1950, as duly qualified medical practitioners: Harper, Ian Thomas, M.B., B.S., 1943 (Univ. Melbourne); Jones, John Morrison, M.B., B.S., 1943 (Univ. Melbourne); Edwards, Cecil Albert Thomas, M.B., B.S., 1946 (Univ. Melbourne); Jacobson, Samuel, M.B., B.Ch., 1937 (Univ. Witwatersrand); O'Donoghue, James, M.B., Ch.B., 1948 (Univ. Liverpool); Shepherd, Ian Leslie, M.B., Ch.B., 1944 (Univ. Aberdeen); Wilkin, James Ernest, L.A.H. (Dublin), 1938.

The following additional qualifications have been registered: Gill, Robert Chalmers (M.B., B.S., 1937, Univ. Sydney, M.R.C.O.G., 1947)—M.D., 1953 (Univ. Sydney); Wilson, Francis Henry Hales (M.B., 1928, Univ. Sydney, M.R.A.C.P., 1946)—F.R.A.C.P., 1953; White, Mervyn McAuley (M.B., B.S., 1935, Univ. Sydney)—M.R.C.O.G., 1947; Eakin, Reay Ignatius (M.B., B.S., 1940, Univ. Sydney)—M.R.A.C.P., 1946, M.R.C.P. (London), 1950; Marsden, Hugh Ernest (M.B., B.S., 1941, Univ. Sydney)—F.R.C.S. (England), 1952; Matchett, Victor Lewin (M.B., B.S., 1945, Univ. Sydney)—D.P.M., 1953 (Univ. Melbourne); O'Mara, Maxwell Lachlan (M.B., B.S., 1945, Univ. Sydney)—D.L.O., R.C.P. and S. (England), 1949, F.R.C.S. (England), 1951.

Nominations and Elections.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Lindsay, Norman Leslie, M.B., B.S., 1947 (Univ. Sydney), 2 Greycairn Place, Edgecliff Road, Woollahra, New South Wales.

The undermentioned have been elected as members of the South Australian Branch of the British Medical Association: Helme, Alan Crichton, M.R.C.S., L.R.C.P. (London), 1952; Riddell, Frank Scoular, M.B., B.S., 1953 (Univ. Adelaide) (qualified 1952); Earl, John Alfred, M.B., B.S., 1949 (Univ. Adelaide); Pulsford, Margaret Jean Bronte, M.B., B.S., 1940 (Univ. Sydney); Richardson, John Patrick, M.B., B.S., 1952 (Univ. Adelaide) (qualified 1951).

Deaths.

THE following deaths have been announced:

KILGOUR.—Donald William Kilgour, on July 7, 1953, at Hillston, New South Wales.

HARBISON.—David Thomas Harbison, on July 13, 1953, at Bowral, New South Wales.

SHAND.—John Cappie Shand, on July 15, 1953, at Sydney.

Diary for the Month.

JULY 28.—New South Wales Branch, B.M.A.: Ethics Committee.
JULY 30.—New South Wales Branch, B.M.A.: Branch Meeting.
JULY 30.—South Australian Branch, B.M.A.: Scientific Meeting.
AUG. 4.—New South Wales Branch, B.M.A.: Organization and Science Committee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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